CHAPTER 51

Traumatic Disorders

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Disorders due to head, spine, and peripheral nerve injuries are among the most commonly seen by neurologists. The impact of these injuries can be devastating for individuals and society, because injuries occurring in a fraction of time can result in death or lifelong impairments associated with chronic pain syndromes. Motor vehicle accidents continue to be a leading cause of neurological trauma despite efforts at primary prevention. Although some neurological injuries such as those due to gunshot and stab wounds reflect a violent society, others, like a lightning strike, are random and capricious. Basic and clinical neuroscience research is directed toward prevention and better treatment of traumatic neurological injury.

ACUTE HEAD INJURY

PATHOGENESIS AND PATHOPHYSIOLOGY. Head injury (HI) accounts for a disproportionate share of morbidity and death in traumatized patients. The past two decades have witnessed a significant decline, however, in overall HI mortality rate from the mid-30% range in the 1970s to less than 20% in the 1990s. This improvement has paralleled an understanding of the secondary injury process and an appreciation that all neurological damage does not occur at the moment of insult but evolves over the ensuing hours and days from various biochemical and molecular derangements. This understanding has led to the development of aggressive treatment strategies to prevent intracranial pressure (ICP) elevations and ischemia. This understanding has led also to extensive clinical therapeutic trials to identify pharmacological neuroprotective agents. The structural injury to the brain at the time of impact is rarely the sole determinant of outcome. Hypoxia, hypotension, mass lesions, increased ICP, ischemia, free radical production, excitotoxicity, and loss of calcium homeostasis are also important.

The frequent occurrence and deleterious effects of the secondary insults of hypoxia and hypotension have long been known.[1,2] Mortality rate is doubled when these insults are superimposed on severe HI (Fig. 51-1), and similarly, concomitant elevated ICP increases morbidity rate.[3] Considerable research has focused on the biochemical and molecular mechanisms of secondary injury. Although these events are multifactorial and interrelated, their timing sequence justifies consideration of each component (Fig. 51-2).

The contribution of oxygen free radicals to secondary injury following HI is a subject of active research.[4,5] Free radicals generated by HI, including superoxide, hydroxyl, hydrogen peroxide, singlet oxygen, and nitrous oxide, have the potential to damage proteins and the phospholipid components of cells and organelle membranes. Additionally, extensive membrane depolarization, induced by trauma, allows for a nonselective opening of the voltage-sensitive calcium channels and an abnormal accumulation of calcium within neurons and glia. Such calcium shifts are associated with activation of lipolytic and proteolytic enzymes, protein kinases, protein phosphatases, dissolution of microtubules, and altered gene expression.[6] Another method of abnormal calcium influx is via activation of excitatory amino acid receptors such as glutamate and aspartate. Excitotoxicity occurs in a widespread fashion after trauma, resulting in cell swelling, vacuolization, and death.[7] Concurrent with the rise in intracellular calcium, magnesium levels are often low immediately following head trauma. Magnesium is a physiological antagonist of calcium and simple replacement of magnesium might attenuate secondary brain injury.[8] Several large clinical trials are evaluating this possibility.

Head injury can result in various types of primary injury occurring at the moment of impact, including lacerations of the scalp, skull fractures, cortical contusions and lacerations, diffuse axonal injury, and intracranial hemorrhage. A coup contusion occurs at the site of impact in the absence of a fracture. A contrecoup contusion occurs in the brain diametrically opposite the point of impact. Acceleration/deceleration forces may cause tearing of nerve fibers at the moment of impact, which is called *shearing injury* or *diffuse axonal injury*. Types of intracranial hematomas include extradural, subarachnoid, subdural, and intracerebral hematomas.

EPIDEMIOLOGY AND RISK FACTORS. The Centers for Disease Control and Prevention estimate that annually

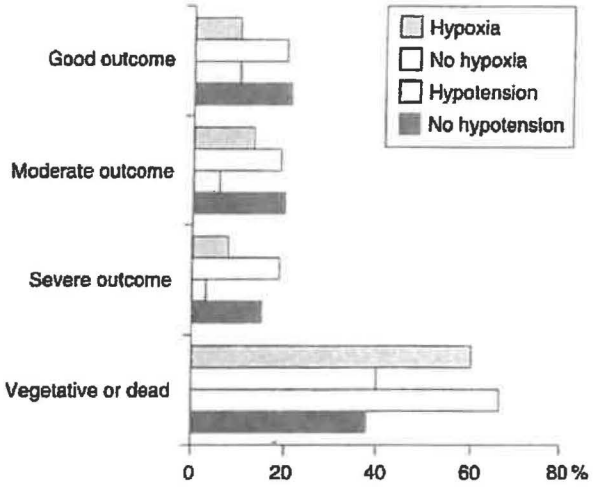

Figure 51-1. Influence of hypoxia and hypotension on mortality after head injury.

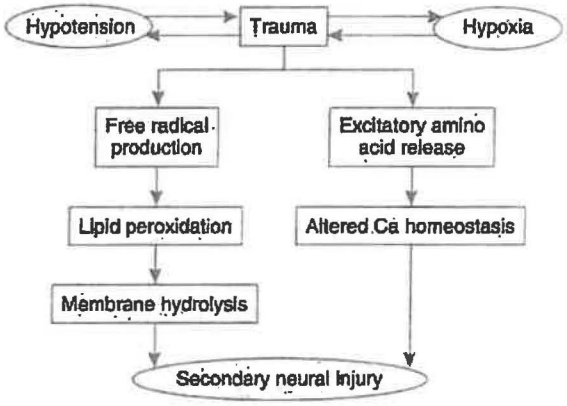

Figure 51-2. Biochemical/molecular substrates of the secondary injury cascade. (From Salaman M: Current Techniques in Neurosurgery, 2nd ed. Philadelphia, Current Medicine, 1993.)

about 1.4 million people sustain a traumatic brain injury (TBI), resulting in 235,000 hospitalizations and 50,000 deaths. Among children ages 0 to 14 years, TBI results in 435,000 emergency department visits, 37,000 hospitalizations, and 2685 deaths.[9] Another 10,000 people sustain spinal cord injuries each year. The peak incidence occurs in men 15 to 24 years of age. Whereas motor vehicle accidents still account for the majority of injuries, the increasing mandate for seat belt use and availability of air bags appear to be reducing injuries. The incidence of penetrating HI from gunshot wounds is ever increasing, and in some urban communities it is now the most common type of injury seen.

A number of classification schemes have been developed for assessment of neurological damage after HI. The most reliable scheme, from a clinical and prognostic standpoint, is based on the Glasgow Coma Scale (GCS):

GCS 3–8: Severe
GCS 9–13: Moderate
GCS 14–15: Mild

Mild HI is termed a *concussion*. Concussions are infrequently associated with structural brain injury and rarely lead to significant long-term sequelae. The postconcussion syndrome is distinct. Attention to neuropsychological testing has shown the possibility of significant cognitive disability following a concussion.[10] Moderate HI may be associated with significant structural injury such as hemorrhage or contusion, but death is uncommon. Severe HI generally results in some form of cognitive or physical disability or in death, especially with very low GCS scores.

The accurate classification of HI is critical in both triage and management. Severe HI should be treated at facilities with appropriate neurotrauma resources, whereas moderate HI requires close clinical observation for possible neurological deterioration. In the setting of mild HI, the most important management issues are related to the need for computed tomographic (CT) scanning, possible inpatient observation, and appropriate follow-up.

CLINICAL FEATURES AND ASSOCIATED DISORDERS. HI is frequently associated with skull fracture, and the significance of the fracture depends on the type of head injury. When associated with severe HI, linear fractures have little practical significance, whereas with mild HI, the presence of a skull fracture increases the risk of an intracranial abnormality by as much as fourfold.[11] Basilar skull fractures may be complicated by cerebrospinal fluid (CSF) leak, infection, and cranial nerve palsies. Patients with basilar skull fractures generally deserve closer clinical monitoring than those with linear skull fracture. In infants and young children, however, linear fractures may be complicated by leptomeningeal cysts or "growing" skull fractures. When such lesions occur, a mass may develop over the fracture site as CSF collects from a disruption of arachnoid intregrity underlying the injury. Cysts always require surgical correction. It is important to follow young children for several months for this possible occurrence.

Infrequently, blunt trauma to the head can tear the middle meningeal artery, diploic veins, or the dural sinuses resulting in a collection of blood in the epidural space, which is an epidural hematoma. Epidural hematomas are associated with a skull fracture in 85% of cases. They most commonly occur in the temporal region, although they can also occur in other locations such as the frontal or parietal regions or within the posterior fossa. The classically described lucid interval associated with this entity is rarely seen because most patients are unconscious from the time of injury. Examination of the head may reveal a laceration and skull fracture over the region of the hematoma. If uncal herniation (i.e, herniation of the mesial temporal lobe) occurs from mass effect, findings may include an ipsilateral cranial nerve III palsy and contralateral hemiparesis (Video 4, Pronator Drift). An ipsilateral hemiparesis can occur as a result of compression of the contralateral cerebral peduncle against the edge of the tentorium. Most epidural hematomas require immediate surgical intervention; however, there are exceptions, particularly in alert pediatric patients.[12]

Acute subdural hematomas occur in approximately 20% of patients with severe HI. Impact injury to the brain can rupture the parasagittal bridging veins (which drain blood from the surface of the hemisphere into the dural venous sinuses) leading to hematoma formation within

the subdural space. Subdural hematomas can also occur without head injury in patients with a coagulopathy or on anticoagulants. Subdural hematomas can also be due to rupture of cerebral aneurysms. Subdural hematomas are usually located over the hemispheres, although other locations, such as between the occipital lobe and tentorium cerebelli or between the temporal lobe and base of the skull, can occur. About 50% of patients with acute subdural hematomas are unconscious from the time of injury. Focal findings can occur because of herniation, as described for epidural hematomas. Surgery within the first 4 hours from injury is generally associated with improved outcome; however, this basic tenet of HI management has been challenged.[13,14]

A subdural hematoma becomes subacute between 2 and 14 days after the injury when there is a mixture of clotted and fluid blood. A subdural hematoma becomes chronic when the hematoma is filled with fluid more than 14 days after the injury. Most patients with chronic subdural hematomas are late middle age or elderly. Up to 50% of those with chronic subdural hematomas, however, have no history of head injury. Patients with chronic subdural hematomas may have multiple types of presentations such as the following: headache and nausea without focal findings; a slow progression of focal signs; altered mental status, apathy, and unsteady gait; an acute focal neurological deficit; fluctuating neurological deficits that may clear; and seizures. Thus, it is easy to see how the various presentations of chronic subdural hematoma can lead to misdiagnoses such as stroke, transient ischemic attack, brain tumor, depression, and dementia. Although occasionally chronic subdural hematomas can be treated with diuretics and corticosteroids, symptomatic patients generally require surgery.

Cerebral contusions are frequently associated with intracranial mass lesions, although they may occur as a result of the coup-contrecoup phenomenon, especially in the temporal and inferior frontal regions. The clinical presentation depends upon the location and severity of the contusion and whether other lesions are present. Patients with small focal contusions can present with speech or motor deficits, whereas larger contusions can act as a mass lesion. Contusions themselves rarely require surgical intervention; however, they necessitate careful clinical and CT follow-up because they may evolve into delayed post-traumatic intracerebral hematomas.[15]

Diffuse axonal injury (DAI) is a frequent CT and pathological correlate of severe head injury, accounting for about 50% of primary brain injuries. It is a result of rotational acceleration of the brain inducing shear, stretch, and deformation of white matter. DAI is usually associated with a poor outcome.[16] DAI when severe is readily identifiable on CT as multiple punctate hemorrhages, typically in the deep white matter and corpus callosum and occasionally in the brain stem. DAI may also occur as a result of mild head injury and may culminate in subtle types of cognitive deficits.

Approximately 10% to 15% of patients with clinically severe HI have a normal CT scan. In such situations, the possibility of extracranial or intracranial vascular disruptions must be considered, and angiography should be considered. A normal CT scan does not preclude the presence of increased ICP. A repeat scan should also be obtained as often abnormalities can be demonstrated in the latter scan. Occasionally, magnetic resonance imaging (MRI) may demonstrate lesions not appreciated on CT in this setting.

Approximately 5% to 10% of patients with severe HI have an associated spine or spinal cord injury. Initial HI evaluation and management thus require simultaneous evaluation and management for potential spinal injuries. The majority of patients with severe HI have multisystem injury. Possibility of other significant and potentially life-threatening injuries should be evaluated and the proper treatment priorities accordingly established.

Multiple medical complications can also occur after HI.[17] Cardiovascular effects of head injury include neurogenic hypertension and cardiac dysrhythmias. Respiratory complications such as neurogenic pulmonary edema, aspiration pneumonia, and pulmonary emboli usually caused by deep venous thrombosis are rather common. Other complications include disseminated intravascular coagulation, hyponatremia due to the syndrome of inappropriate antidiuretic hormone secretion, diabetes insipidus, and stress gastritis.

DIFFERENTIAL DIAGNOSIS. Many nontraumatic disorders may mimic HI, including drug or alcohol intoxication, metabolic disturbances, and subarachnoid or other types of intracranial hemorrhages. When an intoxicated person falls or becomes involved in a fight and sustains a head injury, the emergency evaluation can be further complicated. Often, a patient presents after an apparent traumatic event that itself was precipitated by primary underlying intracranial pathology. Examples include the following: a stroke that causes loss of control while driving; a generalized seizure resulting in a head injury (Video 77, Clonic Seizure); a gait disturbance (due, for example, to Parkinson's disease, diabetes with a sensory ataxia [Video 5, Sensory Ataxia], or cervical spondylitic myelopathy) causing a fall and an HI; and delirium or dementia as a cause of the fall. The physician should not only treat the injury, but should also try to find out if there is an underlying cause.

EVALUATION. The primary evaluation of a patient with HI necessitates a careful neurological evaluation. Because a significant number of these patients have multiple types of trauma, however, systemic evaluation and stabilization take precedence. The ABCs of trauma resuscitation must be followed. As noted previously, correction of hypoxia and hypotension has a significant impact on outcome from HI.

The neurological evaluation of these patients is often complicated by many physical and iatrogenic factors. In comatose patients, a cervical spine series should be performed to exclude fractures before checking the oculocephalic reflex. Severe ocular trauma may obviate examination of pupillary reflexes. Alcohol intoxication and the use of sedatives or muscle relaxants to facilitate patient transport adversely affect the trauma-related neurological findings. Extremity fractures limit the extent of the neurological examination.

The GCS score has been the most reliable indicator of the severity of injury and deterioration or improvement. The initial GCS score often influences early treatment decisions. Thus, an intimate familiarity with the examination essentials to establish the GCS score is important: eye opening, verbal response, and motor response (Table 51-1).[18]

TABLE 51-1

Glasgow Coma Scale

EVALUATION	SCORE
Eye opening	
Spontaneous	4
To voice	3
To pain	2
None	1
Verbal response	
Oriented	5
Confused	4
Inappropriate	3
Incomprehensible	2
None	1
Motor response	
Obeys commands	6
Localizes pain	5
Withdraws	4
Flexion	3
Extension	2
Flaccid	1
Total scare range: 3–15	

Once the GCS score has been established, a more detailed head and neurological examination can be pursued. A careful examination of the head is essential for findings such as scalp lacerations, skull fractures, blood in the external auditory canal, a hemotympanum, or Battle's sign (ecchymosis of the scalp overlying the mastoid bone). Any laterality to the neurological findings (i.e., a dilated pupil, hemiparesis) suggests a focal mass lesion. The higher the GCS score, the more important a detailed mental status examination becomes in determining management. For penetrating HI and especially gunshot wounds, there must also be a full and careful documentation of entry and exit wounds, powder burns, and foreign bodies.

In pediatric patients, the possibility of child abuse must be borne in mind, especially when the primary structural brain injury is subdural hematoma. The *shaken baby syndrome* refers to violent shaking of an infant or small child resulting in retinal hemorrhages and subarachnoid or subdural hemorrhage, but with minimal or no external evidence of severe trauma.[19] When such concerns arise, there must be a careful systemic survey for evidence of other injuries—retinal hemorrhages, skin bruising, new and old long bone fractures.

The more severe the HI, the greater the urgency for CT scanning as an integral part of initial evaluation to exclude lesions that require neurosurgical intervention. The only exception is when other life-threatening injuries take precedence. With minor HI, CT is not routinely indicated. Similarly, if CT is to be obtained, plain skull radiographs are unnecessary. Additional diagnostic evaluations such as MRI, cerebral angiography, and transcranial Doppler examination may be appropriate depending on clinical and CT findings.

MANAGEMENT. The management of minor HI is primarily directed toward a determination of which patients require CT scanning or in-hospital observation. Considerable effort has been expended to determine the risk of deterioration in this group of patients so that appropriate triage protocols may be established. A meta-analysis of a large number of series indicates that approximately 2.5% of patients presenting to an emergency room with minor HI require neurosurgical intervention.[20] This observation has led to current recommendations that only patients with GCS scores of 15 be discharged without prior CT scanning. Risk factors that must be taken into consideration when making this determination include palpable depressed skull fracture, seizures, extremes of age, persistent headache or vomiting, therapeutic anticoagulation, and suspected abuse. Unless there are significant associated extracranial injuries, it is rare for a minor HI to necessitate more than 24 hours of hospital observation.

Moderate HI is generally associated with significant symptoms and findings and necessitates CT scanning and hospitalization for serial neurological monitoring. Repeat CT scanning is indicated if there is any deterioration in neurological functioning. Delayed hematomas are occasionally seen after moderate HI. They usually represent bleeding into areas of contusion not well visualized initially, and patients typically present 24 to 48 hours after injury.[21]

The management of severe HI requires intensive support and monitoring. Because all patients are comatose, intubation and artificial ventilation are mandatory. Blood pressure monitoring and support are crucial to prevent hypotension. Monitoring via Swan-Ganz catheterization may be helpful to maximize perfusion. The first priority is complete and rapid physiological resuscitation. Hypotension (systolic blood pressures less than 90 mmHg) and hypoxia (Pao_2 less than 60 mmHg) should be avoided if possible and corrected if present. Each episode of hypotension and hypoxia cumulatively increases the morbidity and mortality rates from severe head injury, and adequate resuscitation improves outcome from severe head injury.[22]

The institution of ICP monitoring remains somewhat controversial; however, ICP monitors are generally considered appropriate in this setting (GCS 8 or lower) and provide the most reliable guide to treatment of the underlying brain injury. Even patients with severe TBI, with a normal CT scan have a 10% to 15% chance of suffering from elevated intracranial pressures. If they are older than 40 years of age, have unilateral or bilateral posturing or have a systolic blood pressure less than 90 mmHg, then their risk of intracranial hypertension is increased considerably. ICP measurement guides therapy, helps make rational descisions about blood pressure management, and is a strong predictor of outcome. When a ventriculostomy is used to monitor ICP, then a therapeutic tool also exists to treat elevated ICP. Because elevation of ICP more than 20 mmHg is a significant predictor of a poor outcome, an appropriate threshold to begin aggressive ICP treatment appears to be 20 to 25 mmHg.[3,23]

Considerable interest has been focused on cerebral perfusion pressure (CPP) in treating severe HI.[24] CPP is determined by subtracting the ICP value from mean arterial blood pressure and represents the pressure driving cerebral blood flow. Thus, CPP and cerebral blood flow can be positively affected by either lowering ICP or elevating systemic blood pressure. It is generally believed that active attempts to maintain CPP above 70 mmHg enhance cerebral blood flow to ischemic areas of the brain and improve outcome. In patients with intact autoregulation, increasing the

blood pressure increases cerebral arteriolar tone, decreases intracranial blood volume, and reduces ICP. Even in patients with defective autoregulation, a large majority do not experience significant elevation in ICP when the mean arterial pressure is increased.[25]

The primary treatments for elevated ICP include hyperventilation, diuretics, hypertonic saline, and in extreme circumstances, barbiturates. If a ventriculostomy is in place, the drainage of CSF may have significant therapeutic benefits. Hyperventilation has generally been discarded as a primary therapy for increased ICP because of its potential to cause or exacerbate cerebral ischemia. Cerebral blood flow is low in the first few days after severe TBI and hyperventilation can significantly lower it further. For this reason alone hyperventilation should be avoided in the first 3 to 5 days after severe TBI. Chronic hyperventilation (beyond a few hours) is ineffective because of compensatory changes in CSF restoring the pH toward normal. Hyperventilation may be used for short durations in situations while other measures to control ICP are being initiated. $Paco_2$ levels lower than 30 mmHg are rarely indicated. When extreme hyperventilation is required, methods of monitoring for cerebral ischemia, such as jugular venous oxygen saturation ($SJVo_2$), may provide useful clinical information.[26,27] Recently attempts have been made to correlate cerebral perfusion with noninvasive cerebral oximetry.[28] Mannitol is the primary diuretic used to control ICP. Effective doses range from 0.25 to 1.0 g/kg, which are typically given as intermittent boluses. Care must be taken to prevent hypovolemia and hyperosmolarity when giving frequent boluses.

Hypertonic saline is being increasingly used in the management of increased ICP. The blood-brain barrier is impermeable to sodium ions. Since the concentration of these ions, in blood, is several orders of magnitude higher than protein molecules, serum osmolarity is the prime determinant of the movement of water into the brain. Hypertonic saline increases serum osmolarity and intravascular volume, both reducing ICP and enhancing cerebral perfusion. The usual goal is to maintain a serum osmolarity between 300 and 320 mOsm/L.

High-dose barbiturate therapy is generally reserved for those situations in which ICP becomes refractory to other available therapies.[29] The goal of barbiturate therapy is to put the patient in burst suppression. Approximately 60% of oxygen utilized by the brain is used for the maintainance of electroencephalographic activity. Thus, burst suppression reduces the cerebral metabolic rate by about 60%, thereby reducing the cerebral blood flow and cerebral blood volume. This in turn lowers the ICP and improves perfusion of ischemic areas. Many potential complications, including cardiovascular instability, are associated with barbiturate use, however, and extremely careful monitoring is necessary. Although steroids have been often used to treat severe HI, there are no data to support their use to either control ICP or to affect its long-term outcome.[30] A recent randomized, placebo-controlled trial used either a methylprednisolone infusion or a placebo in the first 48 after TBI and looked at outcomes within 2 weeks of randomization. An increase in the risk of death from all causes was found in the corticosteroid group.[31] The use of steroids in the setting of TBI should be abandoned.

Decompressive craniectomy for intractable brain swelling is an older treatment that has recently received renewed attention. A number of operations have been developed for or applied to decompression of the brain at risk for the sequelae of uncontrollable intracranial hypertension. These procedures include Cushing's subtemporal decompression, temporal lobectomy, and the more widely utilized hemispheric and bifrontal decompressive craniectomies. Recent literature suggests, but does not prove, that decompressive procedures, in the appropriate setting, may decrease mortality rate and improve outcome for these patients.

Research has focused on attempting to identify neuroprotective agents to ameliorate secondary injury and thus improve outcome from severe HI. Two large-scale prospective randomized trials of the free radical scavengers tirilazad mesylate and polyethylene glycol-superoxide dismutase (PEG-SOD) have been reported to show no significant reduction in mortality rate or to improve outcome.[32] Recently completed trials with *N*-methyl-D-aspartate (NMDA)–receptor antagonists in an attempt to limit excitotoxicity have not shown any benefit in the clinical setting; however, final results have yet to be published. A small randomized controlled trial reported by Marion and colleagues demonstrated that moderate hypothermia for 24 hours in patients with severe traumatic brain injury and GCS of 5 to 7 on admission hastened neurological recovery and may have improved the outcome. However, a large, multicenter clinical trial did not bear out these positive findings, actually demonstrating a nonstatistically significant increase in mortality rate in GCS 3–4 patients treated with hypothermia.

PROGNOSIS AND FUTURE PERSPECTIVES. Minor HI is rarely associated with significant long-term sequelae. It is well established, however, that a significant minority of patients suffer chronic disability attributable to neurobehavioral sequelae.[33] The exception to this is sports-related minor HI in which death from the second impact syndrome or cognitive disability from cumulative concussions may occur.

Second impact syndrome refers to malignant cerebral edema resulting from relatively mild head trauma occurring after a recent concussion. The syndrome is believed to be due to impaired cerebral autoregulation. It has been well documented that fatal brain swelling may occur in the setting of one minor HI followed in short order by a second minor HI in athletes who are still symptomatic from the first injury.[34] The cumulative effects of multiple minor HIs is recognized in boxing as the *punch drunk syndrome*; however, the occurrence of this syndrome in association with other sports is controversial.

For sports-related minor HI, the American Academy of Neurology has defined three grades of concussion and recommended guidelines for return to play.[35] For grades 1 and 2, there is transient confusion and no loss of consciousness. Resolution of concussion symptoms or mental status abnormalities occurs in less than 15 minutes in grade 1 and more than 15 minutes in grade 2. Grade 3 concussion is any loss of consciousness. Players with grade 1 concussion may return to play the same day if they have a normal sideline neurological assessment including a detailed mental status examination. Players with grades 2 and 3 concussions should not return to play the same day. Guidelines for

TABLE 51-2

When to Return to Play After Removal from Contest

GRADE OF CONCUSSION	TIME UNTIL RETURN TO PLAY*
Multiple grade 1 concussions	1 week
Grade 2 concussion	1 week
Multiple grade 2 concussions	2 weeks
Grade 3—brief loss of consciousness (seconds)	1 week
Grade 3—prolonged loss of consciousness (minutes)	2 weeks
Multiple grade 3 concussions	1 month or longer, based on clinical decision of evaluating physician

*Only after being asymptomatic with normal neurological assessment at rest and with exercise.

From Practice parameter: The management of concussion in sports (summary statement). Report of the Quality Standards Subcommittee. Neurology 1997;48:584.

return to play after removal from the contest (Table 51-2) are necessary because of concern over the cumulative effects of even mild HI and the rare occurrence of second impact syndrome.[36]

Dementia, long recognized as a sequela of multiple head injuries in boxing, was termed *punch drunk* by Martland in 1928 and *dementia pugilistica* by Millspauch in 1937. Neuropathological study of brains of boxers with dementia pugilistica demonstrate β-amyloid protein–containing diffuse plaques and neurofibrillary tangles, which are pathological features of Alzheimer's disease. Increased expression of β-amyloid precursor protein is part of an acute-phase response to neuronal injury, which can lead to deposition of β-amyloid protein.[37]

Patients with moderate HI usually experience both cognitive and physical disabilities and typically require rehabilitation services after acute hospitalization. Nevertheless, the incidence of severe long-term disability is small.

In contrast, few patients with severe HI recover completely to their preinjury state. The most common system to rate recovery is the Glasgow Outcome Scale: good recovery (GR), moderate disability (MD), severe disability (SD), vegetative (V), and death (D).[38] The tirilazad mesylate and PEG-SOD trials provide excellent information on current severe HI outcomes (Table 51-3).

The most active area of research in head injury today is in the field of molecular genetics. It has been noted that certain genes are upregulated, whereas others are downregulated, after both trauma and ischemia. Particular attention has been focused on the apolipoprotein E gene and its various alleles. Certain alleles have been associated with an increased susceptibility and severity of head injury, and others have been linked to improved recoveries after head injury.[39,40] Considerably more research will be required to more completely define and ultimately understand and possibly clinically manipulate these "head injury" genes.

TABLE 51-3

Current Prognosis from Severe Head Injury

	OUTCOMES				
Study	Good Recovery (%)	Moderate Disability (%)	Severe Disability (%)	Vegetative (%)	Death (%)
Tirilazad (n = 557)	44.8	19.6	14.4	21.4	—
PEG-SOD (n = 162)	27	19	20	9	25

Data from Young B, Runge JW, Waxman KJ, et al: Effects of pegorgotein on neurologic outcome of patients with severe head injury. JAMA 1996;276:538–543.

POSTCONCUSSION SYNDROME

Sequelae of mild HI have been recognized for the past few hundred years.[41] In 1866, Erichsen initiated the controversy with his description of patients with persistent complaints after mild head and neck injury due to "molecular disarrangement" of the spinal cord. This condition was known as *railway spine* because many cases were the result of railway accidents. Rigler introduced the concept of compensation neurosis in 1879, because he believed that many persons with persistent symptoms were trying to obtain financial compensation for the injuries. Charcot believed that the impairment was actually the result of hysteria and neurasthenia. Similar debates about the organicity of persistent complaints after mild head injury are still common today, especially in medicolegal cases. The use of the term *postconcussion syndrome* dates back to 1934.

PATHOGENESIS AND PATHOPHYSIOLOGY. Mild head injury may result in cortical contusions due to coup and contrecoup injuries and diffuse axonal injury resulting from sheer and tensile strain damage. Subdural and epidural hematomas can also occasionally result. Release of excitatory neurotransmitters, including acetylcholine, glutamate, and aspartate, may be a neurochemical substrate for mild HI.

EPIDEMIOLOGY AND RISK FACTORS. Mild HI accounts for 75% or more of all brain injuries. The annual incidence of mild HI in the United States is about 140 in 100,000 population, with the relative causes as follows: motor vehicle accidents, 45%; falls, 30%; occupational accidents, 10%; recreational accidents, 10%; and assaults, 5%. Falls are the more likely cause in the elderly, whereas motor vehicle accidents are more common in the young. Men are more commonly injured, with a 2:1 ratio, and about one half of all patients are between the ages of 15 and 34 years. Between 20% and 40% of persons with mild HI in the United States do not seek treatment. Approximately 50% of patients with mild HI develop the postconcussion syndrome.[42]

CLINICAL FEATURES AND ASSOCIATED DISORDERS. The postconcussion syndrome, which is usually the result of mild head trauma, comprises one or more of a large array of symptoms and signs (Table 51-4).[43,44] The most common complaints are headaches, dizziness, fatigue, irritability, anxiety, insomnia, loss of concentration and memory, and noise sensitivity (Video 104, Sound-Induced Vertigo). Loss of consciousness does not have to occur for the postconcussion syndrome to develop. Headaches have been estimated to occur in 30% to 90% of patients who are symptomatic after mild injury. Headaches may occur more often and with longer duration in patients with mild rather than severe HI. Tension-type headaches account for about 85% of all post-traumatic headaches.

TABLE 51-4

Sequelae of Mild Head Injury

Headache Types and Causes
Muscle contraction or tension type
Cranial myofascial injury
Secondary to neck injury (cervicogenic)
Myofascial injury
Intervertebral discs
Cervical spondylosis
C2–C3 facet joint (third occipital headache)
Secondary to temporomandibular joint injury
Migraine
Without and with aura
Footballer's migraine
Greater and lesser occipital neuralgia
Mixed
Cluster
Supraorbital and infraorbital neuralgia
Due to scalp lacerations or local trauma
Dysautonomic cephalgia
Low cerebrospinal fluid pressure syndrome
Hemicrania continua
Chronic paroxysmal hemicrania
Short-lasting, unilateral, neuralgiform headache attacks with conjunctional injection and tearing (SUNCT)
Carotid or vertebral artery dissection
Subdural or epidural hematomas
Hemorrhagic cortical contusions
Cranial Nerve Symptoms and Signs
Dizziness
Vertigo
Tinnitus
Hearing loss
Blurred vision
Diplopia
Convergence insufficiency
Light and noise sensitivity
Diminished taste and smell
Psychological and Somatic Complaints
Irritability
Anxiety
Depression
Personality change
Fatigue
Sleep disturbance
Decreased libido
Decreased appetite
Cognitive Impairment
Memory dysfunction
Impaired concentration and attention
Slowing of reaction time
Slowing of information processing speed
Rare Sequelae
Subdural and epidural hematomas
Cerebral venous thrombosis
Second impact syndrome
Seizures
Nonepileptic post-traumatic seizures
Transient global amnesia
Tremor
Dystonia

Modified from Evans RW: Post-concussion syndrome. In Evans RW, Baskin DS, Yatsu FM (eds): Prognosis of Neurological Disorders, 2nd ed. New York, Oxford University Press, 2000.

Neck injuries, which often occur at the time of the HI, may cause referred headaches due to myofascial, intervertebral disc, and facet joint injury. Greater occipital neuralgia may occur from a direct blow to the nerve or may be associated with muscle spasm of the superior trapezius and semispinalis capitis muscles in the suboccipital region. Similar headaches may arise from the C2–C3 facet joint and are known as *third occipital headaches*. Temporomandibular joint injury may also cause tension-type headaches.

Migraine headaches with and without aura can develop in the hours to weeks after a mild HI. Immediately after mild HI in sports such as soccer, football, rugby, and boxing, children, adolescents, and young adults may have a first-time migraine with aura. This syndrome may be triggered multiple times after additional mild HI and has been termed *footballer's migraine*. Cluster headaches can rarely develop after mild HI. Various other less common causes of post-traumatic headaches are found, including supraorbital and infraorbital neuralgia, dysesthesias over scalp lacerations, carotid or vertebral artery dissections, and subdural and epidural hematomas (see Table 51-1). Subdural hematomas can result in headaches that are nonspecific and that can be mild to severe, paroxysmal or constant, and bilateral or unilateral.

Cranial nerve symptoms and signs also occur. About one half of patients after mild HI report dizziness, which can be caused by various types of central and peripheral pathology, including labyrinthine concussion, benign positional vertigo, and brain stem injury. Blurred vision, reported by 14% of patients, is usually caused by convergence insufficiency. Blurred vision is occasionally the result of cranial nerve III, IV, and VI palsies (Video 32, Cranial Nerve III Palsy). Decreased smell and taste are reported by 5% of patients after mild HI; the symptoms can be due to damage to the olfactory filaments. Approximately 10% of patients describe light and noise sensitivity after mild HI.

Nonspecific psychological symptoms such as personality change, irritability, anxiety, and depression are reported by over one half of patients within 3 months of mild HI. Fatigue and disruption of sleep patterns are also often reported. Post-traumatic stress disorder, which has many symptoms similar to those of the postconcussion syndrome, may occur after mild HI.

Four weeks after a mild HI, 20% of patients complain of impaired memory and concentration. Neuropsychological testing has documented cognitive impairments, including a reduction in information processing speed, attention, reaction time, and memory for new information.

DIFFERENTIAL DIAGNOSIS. The incidence of complications that require neurosurgical consultation after mild HI has been estimated to be between 1% and 3%. For adults with mild HI and an initial GCS score of 13 to 15, the incidence of subdural hematomas is approximately 1% and epidural hematomas about 0.5%.

EVALUATION. Single photon emission computed tomography and positron-emission tomography (PET) scans, brain stem auditory evoked potential studies, and brain mapping currently lack adequate sensitivity and specificity to justify use in the evaluation of the postconcussion syndrome. Electroencephalographic (EEG) studies are usually not indicated except for evaluating post-traumatic seizure disorders. Neuropsychological testing can be quite useful for the evaluation of patients with persistent cognitive complaints.

MANAGEMENT. Treatment should be individualized after the patient's particular problems are diagnosed. Tension- and migraine-type headaches can be treated with the usual prophylactic and symptomatic medications. Greater occipital neuralgia may improve with local anesthetic nerve blocks, which can be combined with an injectable corticosteroid. Physical therapy and transcutaneous electrical nerve stimulators (TENS units) may also help tension-type headaches. For patients with cognitive difficulties, cognitive retraining might be helpful, although the efficacy has not been established by prospective studies. Patients with prominent psychological symptoms may benefit from supportive psychotherapy and use of antidepressant and antianxiety-type medications. Simple reassurance is often the major treatment because most patients improve after 3 months. One of the most important roles for the physician is education of the patient and family members, other physicians, and, when appropriate, employers, attorneys, and representatives of insurance companies.

PROGNOSIS AND FUTURE PERSPECTIVES. The probability of having persistent symptoms and neuropsychological deficits is the same whether a patient is only dazed or loses consciousness for less than 1 hour.[45] Persistent symptoms occur more often in women than men, in patients over the age of 40 years, and in those with a prior history of head trauma. Two years after the injury, about 20% of patients still complain of headaches. Cognitive deficits usually resolve within 3 months after the injury, although a small minority of patients report persistent problems for months or years. When patients have unusual or persistent complaints, the possible contributions of personality disorders, psychosocial problems, or secondary gain should be considered.

A compensation case or lawsuit is often filed in circumstances in which another party may be responsible for the HI, such as a motor vehicle accident or on-the-job injury. In this circumstance, when patients have persistent complaints, many physicians are appropriately concerned about compensation neurosis or malingering being the cause. Patients with claims, however, have similar symptoms that improve with time and similar cognitive test results as those without claims. For many claimants, the end of litigation does not mean the end of symptoms or return to work. They are not cured by a verdict.

CRANIAL NEUROPATHIES

Blunt trauma can result in a stretching injury of cranial nerves, which, when severe, can result in nerves being torn loose from the brain stem. Stretching injuries often occur at points of attachment or at points of angulation. Skull fractures and penetrating trauma such as gunshot wounds can cause nerve lacerations.

Blunt trauma most often damages cranial nerves I, VII, and VIII; less often II, III, IV, and VI (Video 1, Cranial Nerve VI Palsy); and least often V, IX, X, XI, and XII (Video 45, Tongue Atrophy). The incidence of cranial neuropathies increases with more severe HI. For example, the overall incidence of anosmia is about 7% but increases to 30% in patients with anterior fossa fractures or severe HI (Video 230, Exotropia). Even trivial HI can result in anosmia. Injury to the optic nerves and chiasm has been reported in up to 5% of patients with HI. Ocular motility disorders occur in up to one third of patients sustaining closed HI; the abducens nerve is the most commonly injured. Trigeminal nerve and branch injuries often occur as a result of facial trauma (Video 106, Trigeminal Neuralgia). Facial nerve injuries frequently occur with temporal bone fractures.

The site of likely trauma, clinical features, evaluation, management, and prognosis are summarized in Table 51-5.

POST-TRAUMATIC EPILEPSY

PATHOGENESIS AND PATHOPHYSIOLOGY. Post-traumatic seizures may be associated with the typical pathological changes that may be seen in brain injuries, including reactive gliosis, axon retraction balls, wallerian degeneration, microglial scar formation, and cystic white matter lesions.[46] When a contusion or cortical laceration is present, the breakdown of hemoglobin releases iron. Based on animal and cell culture studies, iron may increase intracellular calcium oscillation and may increase free radical formation through activation of the arachidonic acid cascade, producing increased intracellular calcium resulting in excitotoxic damage, neuronal death, and glial scarring, which lead to epileptiform activity.[47] These findings suggest a possible role for neuroprotective treatment in the future. There is also evidence suggesting that post-traumatic seizures may be a result of alterations of intrinsic membrane properties of pyramidal neurons together with enhanced NMDA synaptic conductances.[48]

EPIDEMIOLOGY AND RISK FACTORS. Head injury is the cause of about 4% of all cases of epilepsy. In teenagers and young adults, HI is the most common cause of symptomatic epilepsy. Post-traumatic seizures can be divided into different types based on the time of onset after the injury. Immediate seizures occur within minutes of the injury. Early seizures occur within 1 week, whereas late seizures occur after 1 week.

The incidence of early post-traumatic seizures is about 4%. Early seizure rates are higher in patients with more severe HI, intracranial and subdural hematomas, depressed skull fractures, focal neurological signs, loss of consciousness, or post-traumatic amnesia present for more than 24 hours, and in children under the age of 5 years.

Late seizures have an incidence of about 2%.[47] Risk factors for late seizures include penetrating missile wounds, an early seizure; intracerebral, epidural, and subdural hematomas; GCS score of 10 or less; depressed and linear skull fractures; and cortical contusions.

CLINICAL FEATURES AND ASSOCIATED DISORDERS. About one third of early seizures occur during each of the following times: within the first hour, between 1 and 24 hours, and between 1 and 7 days. Over 60% of early seizures are partial, whereas the others are generalized tonic-clonic (Video 74, Secondary Generalized Seizure; Video 79, Tonic/Atonic Seizure). About 10% of patients develop status epilepticus. Immediate concussive convulsions are not an epileptic phenomenon but instead are due to a brief traumatic functional decerebration resulting from loss of cortical inhibition.[49] Antiepileptic medication is not indicated for concussive convulsions.

TABLE 51-5

A Summary of Traumatic Cranial Neuropathies

CRANIAL NERVE	SITE OF LIKELY TRAUMA	CLINICAL FEATURES	EVALUATION	MANAGEMENT	PROGNOSIS
I	Any part of head. More common with occipital than frontal trauma	Decreased or absent sense of smell	Check for possible ethmoid fractures, CSF rhinorrhea, and injury to orbital surface of frontal lobes	Educate patient	Reso in up to 50% of cases, usually during first 3 months but up to 5 years after injury
II	Intracanalicular often associated with a skull base fracture	Decreased or loss of vision. Scotomas, sector, and altitudinal defects can occur	Check visual acuity, fields, and for afferent pupillary defect. CT scan and ultrasound A and B are useful to assess for possible compressive lesions	Controversial for indirect optic neuropathy: include observation, high-dose steroids, and surgery. Surgical decompression often recommended in cases of delayed onset of visual loss	Extremely variable; from 0–100% depending upon the study
III	Where the nerve enters the dura at the posterior end of the cavernous sinus. Uncal herniation much less common	Dilated pupil and turned out eye present in complete palsy. Anisocoria and diplopia in partial lesions	CT and/or MRI scans to look for a compressive lesion	Symptomatic such as eyepatch, semitransparent tape to glasses and prisms for diplopia. Muscle shortening procedures may be helpful for permanent diplopia	Recovery begins within 2–3 months when nerve in continuity. Aberrant regeneration often occurs with findings such as lid elevation or pupillary constriction with attempted adduction or depression
IV	Stretching or contusion of the nerve as it exits the dorsal midbrain near the anterior medullary velum	Trauma is the most common cause of trochlear palsies. Bilateral palsies are rather common	Same as above	Same as above	Only 50% recover because of frequent avulsion of the trochlear nerve
V	Trigeminal nerves and branches are commonly injured with facial trauma, especially supraorbital and supratrochlear nerves. The infraorbital nerve is often injured in orbital floor blowout fractures	Injury to the gasserian ganglion and trigeminal trunk is rare after closed head trauma	Imaging studies to exclude underlying fractures	Decompression of the infraorbital nerve in orbital floor fractures. Symptomatic for hyperpathia due to supra- and infraorbital neuropathies with medications such as carbamazepine, tricyclics, and baclofen	Hyperpathia in the distribution of the nerve may be permanent
VI	As the nerve ascends the clivus, in fractures of the petrous bone along with VII and VIII, in the superior orbital fissure along with III and IV, and in its subarachnoid course due to raised intracranial pressure	Bilateral palsies are rather common	Same as above	Same as above	Recovery often occurs after 4 months
VII	Most commonly within the petrous bone but can be injured anywhere along its course	Injured in 50% with a transverse temporal bone fracture. Facial palsy in 25% with a longitudinal fracture, often with a delayed onset	CT scan to evaluate temporal bone trauma. A nerve conduction study 5 or more days after the injury is helpful to assess the degree of nerve injury	Facial nerve decompression is usually indicated for transverse fractures. Artificial tears and eye patch at night to prevent exposure keratitis	Spontaneous recovery usual after longitudinal fractures. When due to transverse fractures, 50% recovery after decompression

Continued

TABLE 51-5

A Summary of Traumatic Cranial Neuropathies—cont'd

CRANIAL NERVE	SITE OF LIKELY TRAUMA	CLINICAL FEATURES	EVALUATION	MANAGEMENT	PROGNOSIS
VIII	Labyrinthine concussion without a skull fracture is the most common site. Conductive hearing loss follows longitudinal temporal bone fractures in over 50% of cases. Transverse fractures result in vestibular and cochlear nerve laceration in over 80%	Findings associated with a fracture of the petrous portion of the temporal bone include hemotympanum or tympanic membrane perforation with blood in the external canal, hearing loss, vestibular dysfunction, peripheral facial nerve palsy, CSF otorrhea, and ecchymosis of the scalp over the mastoid bone (Battle's sign). Benign positional vertigo occurs in about 25% of patients following head trauma	Examine the external canals and tympanic membranes. An audiogram including pure-tone and speech audiometry, acoustic reflexes, and middle ear function is used to assess hearing loss. An electronystagmogram (ENG) is used to assess vestibular function. CT scan to evaluate temporal bone trauma	A hearing aid may help those with sensorineural hearing loss. Surgical correction is indicated for conductive hearing loss due to ossicular chain disruption. Positioning maneuvers, such as Epley's or Semont's, which move the debris out of the semicircular canal and into the utricle, can be curative for benign positional vertigo	Following temporal bone fractures, patients with low-or high-frequency hearing loss may have some recovery, but those with low- and high-frequency loss usually do not recover. Vertigo due to a labyrinth concussion usually resolves within a year
IX, X, XI, XII	Gunshot or stab wounds occasionally cause injury. A fracture of the occipital condyle, Collet-Sicard syndrome, can injure all four nerves. The peripheral portion of XI can be injured in surgical procedures such as posterior cervical lymph node biopsies. The hypoglossal and recurrent laryngeal nerves can be traumatized in anterior neck operations such as carotid endarterectomy	Lower cranial nerve findings associated with signs of brain stem compression are consistent with an intracranial lesion, whereas the presence of a Horner's syndrome is consistent with an extracranial lesion	A careful clinical examination is mandatory. CT and MRI are both useful, depending upon the case	Treatment of Collet-Sicard syndrome is supportive with elevation of the head for drainage of excess saliva and IV or nasogastric nutrition until normal swallowing returns. Accessory nerve injuries in the neck may require exploration with neurolysis or resection and repair or grafting, depending on the degree of injury	Collet-Sicard syndrome may show slow partial recovery. Patients with vagal, spinal accessory, and hypoglossal nerve injuries associated with carotid endarterectomy often recover

About 50% of patients with late seizures have their first seizure within 1 year of the injury and about 80% within 2 years. Over 60% of the seizures are generalized tonic-clonic with or without focal onset, whereas the others are simple or complex partial seizures.

DIFFERENTIAL DIAGNOSIS. In some cases, post-traumatic seizures must be distinguished from disorders that can result in psychogenic nonepileptic events including conversion, factitious behavior, malingering, reinforced behavior pattern, somatization, post-traumatic stress disorder, hypochondriasis, panic disorder, depersonalization, dissociative disorder, and hyperventilation syndrome. Because mild HI uncommonly results in seizures, non-epileptiform events should be considered when these patients have "funny spells," or episodes that are atypical for epilepsy.[50]

EVALUATION. A detailed history from the patient and witnesses, if any, of the event; review of pertinent medical records; and general and neurological examination are often the bases for diagnosing post-traumatic seizures. Although an EEG study is certainly important, epileptiform activity is seen in only 50% of patients. Video or ambulatory EEG studies may be worth while in occasional cases when the diagnosis is unclear. A CT or MRI scan of the brain may also be indicated.

MANAGEMENT. In patients with risk factors for early seizures, prophylactic treatment with phenytoin reduces the incidence of early seizures. After the first week, however, phenytoin can be gradually withdrawn because the incidence of late seizures is not reduced by continuing treatment.[51] Instituting long-term anticonvulsant treatment is beneficial for patients after an initial late seizure.

PROGNOSIS AND FUTURE PERSPECTIVES. The remission rate for post-traumatic epilepsy is about 50%. An EEG is of no specific value in predicting either the development or remission of post-traumatic epilepsy. A greater number of seizures and abnormal findings on neurological examination decrease the chance of remission.

POST-TRAUMATIC MOVEMENT DISORDERS

An association between injuries and involuntary movements has been suggested since the 19th century speculation of Parkinson, Charcot, and Gowers. When a movement disorder begins shortly after a brain injury, a cause-and-effect relationship is apparent. An association is less obvious with longer latencies after the injury and after peripheral trauma where there may be a temporal but not causal relationship.[52] In some cases, the patient may not have noticed or reported a mild movement disorder that was present before the injury.[53,54]

PATHOGENESIS AND PATHOPHYSIOLOGY. Direct trauma to subcortical and substantia nigral neurons can result in movement disorders occurring shortly after an injury. Movement disorders occurring months following the injury have been hypothesized to be related to sprouting, remyelination, ephaptic transmission, inflammatory changes, oxidative reactions, and central synaptic reorganization. Peripheral trauma that precedes the development of a movement disorder may alter sensory input, leading to central cortical and subcortical reorganization.[55,56]

EPIDEMIOLOGY AND RISK FACTORS. Movement disorders due to trauma are rare, and specific epidemiological and risk factor studies have not been performed.

CLINICAL FEATURES AND ASSOCIATED FINDINGS. Although HI does not result in Parkinson's disease with pathological findings of Lewy bodies, HI may result in a temporary exacerbation of motor function in patients with pre-existing Parkinson's disease.[53] Parkinsonism is a rare complication of single closed HI and may also occur after penetrating bullet and knife injuries of the brain stem. Repeated HI as in the case of boxers with dementia pugilistica may result in parkinsonism in association with many other neurological signs. Parkinsonian tremor may rarely be associated with peripheral body trauma, although a direct causal relationship cannot be established.[57]

Postural and kinetic tremor can be due to direct traumatic lesions of the dentatothalamic circuit. Benedikt's syndrome (unilateral cranial nerve III palsy and contralateral ataxic hemiparesis) can be associated with rest and postural and kinetic tremor (Video 14, Tremor with Ataxia). Postural-kinetic tremors of the arms, legs, or head may occur within weeks of mild HI even without loss of consciousness. Peripheral trauma can induce tremor, which can occur along with complex regional pain syndrome, dystonia, and myoclonus (Video 57, Myoclonus).[56,57] Myoclonus, dystonia, and athetosis may be present in patients with post-traumatic tremors.

Contralateral dystonia can be due to a lesion in the striatum, particularly the putamen (Video 242, Post-traumatic Dystonia). Causes include perinatal trauma, closed HI (severe much more often than mild), and thalamotomy. The onset of dystonia may have a latency period from 1 month to 9 years. Spastic dystonia due to pyramidal and extrapyramidal injury and paroxysmal nocturnal dystonia are variants of post-traumatic dystonia. Often patients develop post-traumatic dystonia as a delayed sequela of severe HI, initially characterized by coma and quadriplegia. After the patient awakens and the plegia improves, severe action dystonia develops. Minor or moderate local peripheral trauma can be associated with focal dystonia, sometimes in patients with reflex sympathetic dystrophy. Examples of peripherally induced dystonia include the following: blepharospasm after surgery on the eyelids (Video 47, Blepharospasm); oromandibular dystonia after dental procedures; spasmodic dysphonia after facial injuries (Video 48, Spasmodic Dysphonia); cervical dystonia after neck injuries such as whiplash; and foot dystonia after stubbing a toe.

A report by Kraus and colleagues studied survivors of severe HI admitted to the hospital with GCS of 8 or less. Of the 264 survivors, follow-up was obtained on 221, and 22% reported or showed evidence of movement disorders, half transient and half persistent. Tremor, usually kinetic, and dystonia were the most common disorders and usually developed with a post-trauma latency of 2 to 24 months.

Chorea, choreoathetosis, and ballismus can follow blunt head trauma with injury to the striatum, subthalamic nucleus, and anterior thalamus (Video 18, Limb Chorea). The onset is usually days to months following the trauma.

Action myoclonus, palatal myoclonus, and segmental myoclonus may result from head injury not associated with

anoxia or epilepsy (Video 57, Myoclonus). Segmental myoclonus may also occur after spinal cord injury.

A deep, burning pain followed by persistent, involuntary, and irregular movements of the toes and feet, termed *painful legs and moving toes*, can be associated with minor foot and ankle injuries. Hemifacial spasm has been rarely associated with trauma. Additionally, after amputation, the remaining stump can involuntarily jerk.

DIFFERENTIAL DIAGNOSIS. Because the relationship between many injuries and the movement disorder may be circumstantial and retrospective, an idiopathic movement disorder is always a diagnostic consideration. The possibility of psychogenic movement disorders, which may be more common in women, should also be considered. Because movement disorders often develop several weeks or months after trauma and because patients often receive neuroleptic medications after acute injuries, drug-induced movement disorders are another possible cause.

EVALUATION. Neuroimaging such as MRI and occasionally PET can demonstrate responsible brain lesions. Depending on the case, testing to exclude other potential causes of the movement disorder such as Wilson's disease may also be appropriate.

MANAGEMENT AND PROGNOSIS. Post-traumatic parkinsonism may respond to dopaminergic and cholinergic medications. Post-traumatic action tremors only occasionally respond to standard medical treatment. Selected cases may benefit from botulinum toxin injections into involved muscles and ventrolateral thalamotomy. Medications are usually not helpful for dystonia associated with central and peripheral trauma, although botulinum toxin injections can produce temporary relief.[55] Post-traumatic chorea and choreoathetosis may respond to valproic acid and haloperidol, whereas trauma-induced cortical myoclonus can be treated with clonazepam.

SPINAL CORD INJURY

Over the past decade there have been significant advances in transport, emergency management, pharmacological resuscitation, comprehensive acute care, and rehabilitation. Nevertheless, spinal cord injury (SCI) remains a physically and emotionally devastating problem. The neurologist may thus be confronted with the need for recognition of a potential spinal injury or SCI and institute appropriate stabilization procedures.

PATHOGENESIS AND PATHOPHYSIOLOGY. The pathophysiology of SCI may be divided into two distinct phases—primary and secondary injury. Primary injury refers to the structural damage occurring instantly after the traumatic event. Further primary injury may occur, however, if an injured spine is not adequately immobilized. Secondary injury refers to a pathophysiological cascade initiated shortly after injury, including such insults as ischemia, hypoxia, edema, and various harmful biochemical events. Because it is extremely rare for the primary injury to cause transection of the spinal cord, and it has been shown that less than 10% of the cross-sectional area of the spinal cord supports locomotion, it is very important to focus clinical attention on the secondary injury process.[58]

Ischemia is a very prominent feature of post-SCI events. Within 2 hours of injury there is a significant reduction in spinal cord blood flow. This ischemia may be confounded by loss of the normal autoregulatory response of the spinal cord vasculature. When autoregulation is lost, blood flow becomes dependent on systemic pressures. Thus, in the multitraumatized patient or the patient with vasogenic spinal shock complicating the SCI, severe systemic hypotension may exacerbate the spinal cord ischemia. Hypovolemia and hypotension should be corrected rapidly. Hypotension in particular may increase the area of secondary injury in the spinal cord and worsen outcome. Crystalloids, colloids, and blood products may be used to replace volume as needed. If hypotension persists despite an adequate volume status, vasopressors should be used to maintain a mean arterial pressure of approximately 80 mmHg.

Edema formation is another feature of the secondary injury process. Edema develops first at the injury site and subsequently spreads into adjacent and sometimes distant segments of the cord. The relationship between this edema and worsening of neurological function is not well understood.

Many biochemical mechanisms have been implicated in the evolution of the pathological changes and physiological derangements occurring after SCI. Electrolyte disturbances have been well documented, including increased intracellular calcium level, increased extracellular potassium level, and increased sodium permeability. Other events such as excitatory neurotransmitter accumulation, arachidonic acid release, endogenous opiate activation, and prostaglandin production have all been implicated as damaging elements of the post-injury cascade. Other events, free radical production and lipid peroxidation, are believed to play a central role in this process. Ultimately, however, all of these events cumulatively result in ischemia, edema formation, membrane destruction, cell death, and eventually permanent neurological deficits.

EPIDEMIOLOGY AND RISK FACTORS. Spinal cord injury occurs at a rate of 30 to 40 per 1 million population per year, resulting in approximately 10,000 new cases each year. The prevalence of SCI is over 200,000.[59] SCI occurs primarily in young males 18 to 25 years of age. The primary precipitant of injury is motor vehicle accidents, although in some areas of the country, swimming and diving-related accidents may take precedence. Sports-related injuries account for less than 5% of the total. In some urban areas the incidence of gunshot-related SCI is increasing (Fig. 51-3). SCI rarely occurs in isolation, and over 75% of these patients have some other systemic injury. In 10% to 15%, there is an associated head injury. This concern has led to the widely quoted clinical maxim that all multitraumatized patients or any patient with a severe head injury should be presumed to have a spine injury or SCI until proved otherwise.

CLINICAL FEATURES AND ASSOCIATED DISORDERS. Although general physical assessment of the patient may raise concerns over a possible SCI, a detailed neurological assessment is the only reliable means to rule out this diagnosis. A standardized means of performing and recording the neurological examination has been advocated by the American Spinal Injury Association (ASIA) and has been accepted internationally by both clinicians and researchers (Fig. 51-4). With a complete transverse myelopathy, all

motor and sensory function below the level of injury is absent. The neurological level of injury is the most caudal or lowest spinal cord segment with normal sensation and a muscle strength of 3/5 or better. An incomplete injury is present when there is preservation of any motor or sensory function below the zone of injury, including sacral sparing.

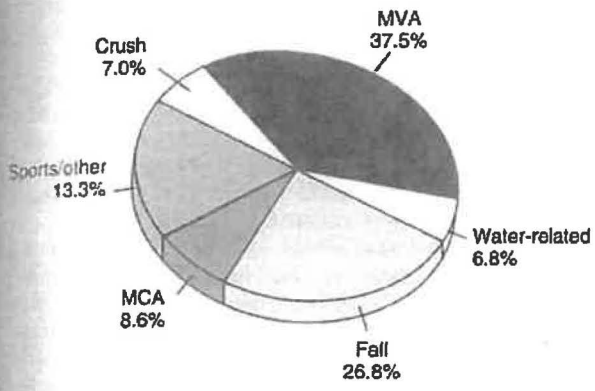

Figure 51-3. Epidemiology of spinal cord injury. MCA, motor cycle accident; MVA, motor vehicle accident.

Apart from weakness, paralysis, and sensory changes, in the acute stage, depending on the level of injury, certain physiological changes are often evident. These changes include spinal shock, paradoxical respiration, low body and high skin temperature, priapism, and a reduced sweating level. Spinal shock is usually seen in injuries above T6 level and is characterized by hypotension, bradycardia, and an absence of motor and sensory function as a result of spinal cord injury. The higher the level of injury and the more complete the injury, the greater the intensity and duration of spinal shock. The motor and sensory functions usually recover (if any recovery is possible) within an hour, but the autonomic component of spinal shock (loss of sympathetic function) may persist for days to months. Paradoxical respiration is the result of diaphragmatic contraction in the face of intercostal muscle paralysis. The skin is warm because of a loss of sympathetically mediated peripheral vasoconstriction; this also causes the core temperature to fall. Priapism may result from an unopposed

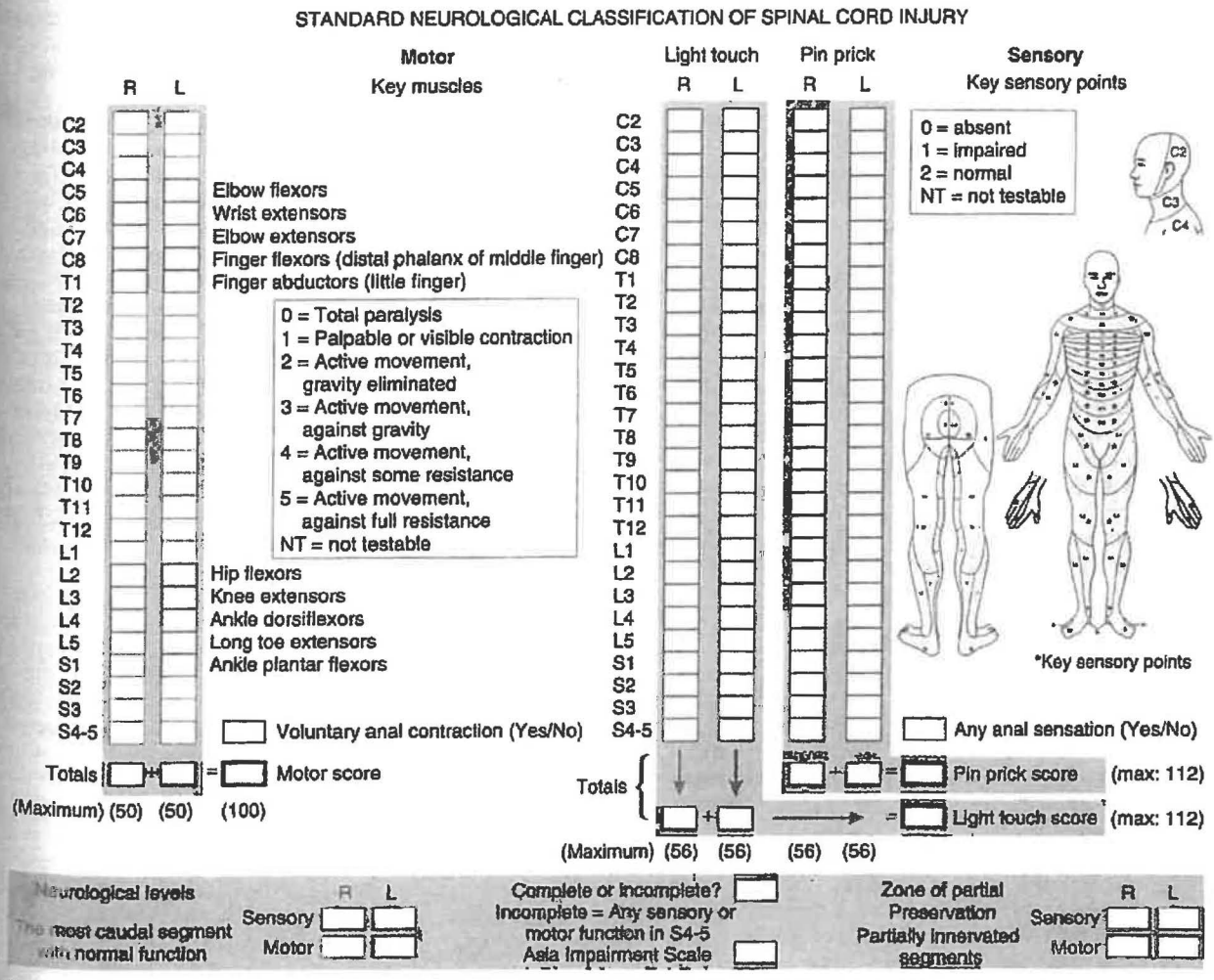

Figure 51-4. The ASIA system for examination and classification of spinal cord injury. (From American Spinal Injury Association: Standards for Neurological and Functional Classification of Spinal Cord Injury, Revised 1992, p 278.)

parasympathetic function, and sweating is lost because of loss of sympathetic activity.

Basic knowledge of myotomes, dermatomes, and deep tendon reflexes is essential for localization. Injury to the upper cervical cord can also damage brain stem structures either because of direct trauma or because of vascular injury of the vertebral arteries. This cervicomedullary syndrome is characterized by respiratory dysfunction, hypotension, variable tetraparesis, hyperesthesia from C1–C4, and sensory loss of the face with an onion-skin pattern.

Partial or incomplete lesions of the spinal cord can result in four patterns of deficit. In the central cervical cord syndrome, the paresis involves the upper extremities, especially the hands, more than the lower extremities. The mechanism of injury is acute cord compression between bony bars or spurs anteriorly and thickened ligamentum flavum posteriorly, resulting in relatively more injury to the medial segments of the corticospinal tracts, which control arm function. The anterior cord syndrome, complete paralysis and hyperesthesia at the level of the lesion but intact light touch and vibration sense, is due to a large disc herniation compressing the anterior cord but without compression of the dorsal columns. The posterior cord syndrome is posterior column damage with impaired light touch and proprioception resulting from hyperextension injuries with fractures of the vertebral arch. The Brown-Séquard syndrome, caused by a lesion of half of the spinal cord, is defined by ipsilateral motor and proprioceptive loss and contralateral pain and temperature loss with the upper level one or two segments below the level of the lesion. This syndrome can occur after various injuries, including penetrating trauma, hyperextension and flexion injuries, locked facets, and compression fractures.

The conus medullaris syndrome is due to a compression injury at T12 that can occur from a disc herniation or a burst fracture of the body of T12. Because almost all the lumbar cord segments are opposite the T12 vertebral body, a severe compression can produce dysfunction in any or all of the lumbar as well as the sacral segments. Flaccid paralysis of the legs and anal sphincter with variable sensory deficits can be present. Because the spinal cord usually terminates at the L1-L2 disc space, trauma below this level injures the nerve roots. The cauda equina syndrome, which is compression of nerve roots below the L1 level, can be caused by fractures and dislocations of the spine or large posterocentral intervertebral disc herniations. Lower motor neuron deficits result with variable sensorimotor, reflex, bladder, bowel, and sexual dysfunction.

In the unconscious patient, neurological evaluation for SCI is very difficult. The only clues to the presence of a significant SCI may be a lack of facial grimacing to peripherally applied painful stimuli, indicating a sensory loss over the trunk, or a lack of withdrawal behavior in the arms or legs in response to painful stimulation applied to the head or face.

The biomechanics of the pediatric spine are fundamentally different from that of the adult. These differences—ligamentous laxity, wedge-shaped vertebrae, horizontally oriented facets—account for distinct clinical presentations. The clinical differences between adult and pediatric SCI include the following: disproportionate involvement of the upper cervical spine in pediatric patients, high frequency of spinal cord injury without radiographic abnormality, high susceptibility to the delayed onset of neurological deficits in children, and a higher proportion of complete neurological injuries in children (Table 51-6).[60,61]

A unique SCI syndrome—the burning hands syndrome—was first described in sports injury.[62] This syndrome appears to be a variation of central cord syndrome associated with severe burning paresthesias and dysesthesias in the hands or the feet. Other signs of neurological dysfunction are minimal or absent. Over 50% of the time there is an underlying spine fracture-dislocation. It is important to differentiate this syndrome from the much more common and usually innocuous "burner" or "stinger" of brachial plexus origin.

The syndrome of neurapraxia is also of special concern after athletic injury. Affected individuals experience dramatic, although transient, neurological deficits including quadriplegia. Frequently this syndrome is associated structurally with degenerative or congenital spinal canal stenosis. Many attempts have been made to quantitate the level of risk to these individuals from continued athletic participation; however, considerable controversy still exists.[63,64]

The majority of spinal cord–injured patients have at least one other system injury. Occasionally these injuries take precedence in evaluation and treatment. If one level of bony injury has been identified, it is necessary to survey the entire spine because there is a 10% to 15% incidence of spine injury at other levels.

Various problems may occur with chronic SCIs. Within a few weeks of the injury, hyperactive reflexes develop, which are associated with hypertonicity of the extremities (Video 80, Hyper-reflexia). Although moderate spasticity is not a problem for the patient, severe spasticity may lead to flexor spasms and contractures (Video 20, Tonic Spasms). Central deafferentation pain, dysesthesias described as burning, stinging, or freezing, may be present. Autonomic dysfunction can occur as early as 2 to 3 weeks after complete or incomplete lesions of the cervical or thoracic cord. This is characterized by massive sympathetic outflow in response to certain noxious stimuli, usually genitourinary manipulations. In severe cases marked elevations in blood pressure, bradycardia, flushing, headache, nausea, and cardiovascular collapse may occur. There is severe vasoconstriction below the level of the cord injury and vasodilatation above it. This unopposed reflex sympathetic activity may be seen after the

TABLE 51-6

Spine and Spinal Cord Injuries

CHARACTERISTICS	ADULT	PEDIATRIC
Mechanism of injury	Motor vehicle accidents	Pedestrian/falls
Level of injury		
C1–C3	1–2%	60%
C3–C7	85%	30–40%
Thoracolumbar	10–15%	5%
Type of injury		
Fracture-dislocation	>70%	25%
Subluxation alone	<20%	>50%
SCIWORA	Rare	Up to 50%
Delayed neurological deficits	Rare	Up to 50%

SCIWORA, spinal cord injury without radiological abnormalities.

period of the spinal shock, and is usually treated by withdrawing the initiating stimulus and administering vasodilators. Spinal anesthesia eliminates this reflex sympathetic outflow and should be considered for patients undergoing genitourinary procedures in the operating room.

Orthostatic hypotension is frequent when the patient is first allowed to sit. Autonomic hyper-reflexia or "mass reflex" may be triggered by noxious stimuli such as fecal impaction, urinary tract infections, distention of the bladder, uterine contraction, and decubiti. Manifestations include nausea, throbbing headache, skin blanching and diaphoresis below the level of the cord lesion, and paroxysmal hypertension.

When patients develop progressive neurological deficits any time from several months to many years after SCI, post-traumatic syringomyelia may be responsible. Present in up to 3% of patients, four kinds of trauma may be responsible: repeated microtrauma, arachnoiditis, severe single trauma, and minor single trauma.[65] One mechanism of syrinx formation is an initial hematomyelia with subsequent resorption and formation of a cyst cavity.

DIFFERENTIAL DIAGNOSIS. Very few disorders mimic acute SCI. Occasionally a patient with hysterical paralysis presents a diagnostic and therapeutic dilemma. It should be borne in mind that true SCI is almost always associated with obvious physiological disturbances such as bradycardia, hypotension, and respiratory compromise.

EVALUATION. The injured spine may become a radiological diagnostic dilemma, and one must constantly bear in mind the normal radiological appearance of the spine. The initial evaluation should always include plain radiographs of the cervical or thoracolumbar spine, depending on the neurological findings.[66–68] If an abnormality is identified, complete spine films must be obtained because there is a 10% to 15% incidence of multiple fractures following trauma.

Additional useful information can often be obtained by performing flexion-extension views. Extreme caution must be taken if instability is suspected, however. To better interpret plain films or to better define a definite abnormality, CT is extremely helpful. MRI is sometimes necessary for the complete evaluation of SCI. MRI is the only diagnostic modality that allows direct visualization of the injured cord.[69]

A number of guidelines have been developed to aid the clinician in clearing the potentially injured spine. The most widely quoted of these are the Eastern Association for the Surgery of Trauma guidelines.[70]

The first principle in evaluating the cervical spine is a clear visualization of all seven cervical vertebrae. At times, a swimmer's view is necessary to visualize this region. If the C7–T1 junction cannot be imaged on plain films, a CT should be performed.

All films should be reviewed for alignment of the vertebral bodies and the facet joints, vertebral body height, and interpedicular distance and normal soft tissue configuration. When evaluating the C1–C2 region, careful attention must be given to the distance separating the anterior arch of C1 and the odontoid process. In an adult, this distance is normally less than 2 mm; in children up to 5 mm distance is acceptable. If any questions arise over possible pathological separation, flexion-extension films may be helpful. Alternatively, MRI may be employed to evaluate the competency of the transverse axial ligament.[71]

Another important consideration when evaluating cervical spine films is the entity of pseudosubluxation. Pseudosubluxation of up to 3 to 4 mm of C2 on C3 may appear in up to 40% of normal radiographs in children younger than 8 years old. A similar abnormality can occur at C3–C4 in up to 20%. If there is any question about this entity, flexion-extension views should be helpful. A true subluxation cannot be reduced, as contrasted with a pseudosubluxation.

The usual mechanism for atlas fractures involves axial compression of the head with downward displacement of the occipital condyles into the lateral masses of C1. The most important radiographical view is the open-mouth odontoid. With a C1 fracture, the lateral masses of C1 are offset laterally with respect to C2. CT scanning clearly demonstrates the associated C1 ring fractures (Fig. 51-5).

Dens fractures occur as a consequence of either forced hyperextension or hyperflexion of the head. With hyperflexion injuries and associated transverse axial ligament damage, the dens is displaced anteriorly with forward subluxation of C1 on C2. With hyperextension injuries the dens is displaced posteriorly with associated posterior subluxation of C1 on C2. Three types of dens fractures may be seen—type 1, fracture line across tip of dens; type 2, at the base of the dens; and type 3, with a fracture extending into the body of C2. CT scanning may not demonstrate a dens injury if the fracture line is axially oriented.

Another type of axis fracture is the hangman's fracture, usually resulting from hyperextension. Typically there are fractures through both pedicles of C2 with associated forward subluxation of C2 on C3 (Fig. 51-6).

Except in infants and young children, over 85% of cervical fractures occur below C3. Locked facets, either unilateral or bilateral, and compression fractures are the most common lesions. These types of injuries are characteristically produced by flexion complicated by axial loading.

Compression fractures are characterized radiographically by loss of vertebral height, retropulsion of bone into

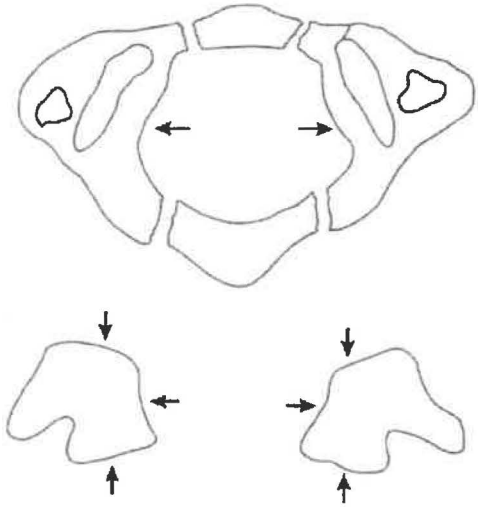

Figure 51-5. A C1 burst or Jefferson's fracture.

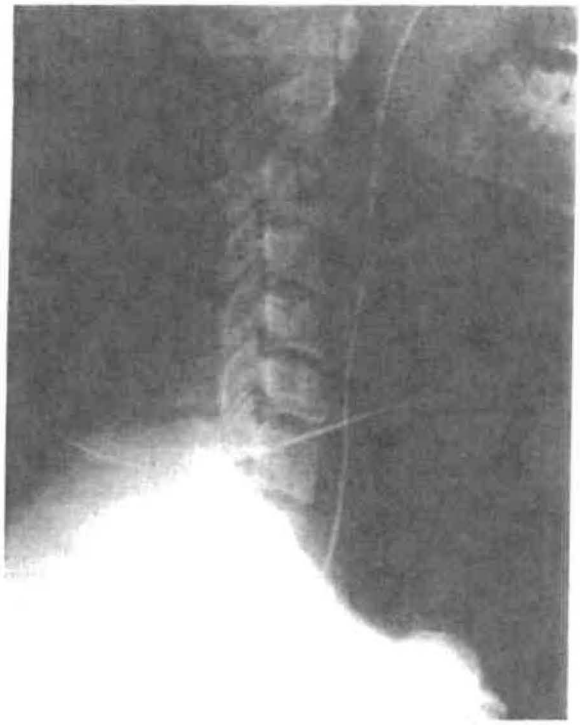

Figure 51-6. Plain radiograph of a typical C2-hangman's fracture.

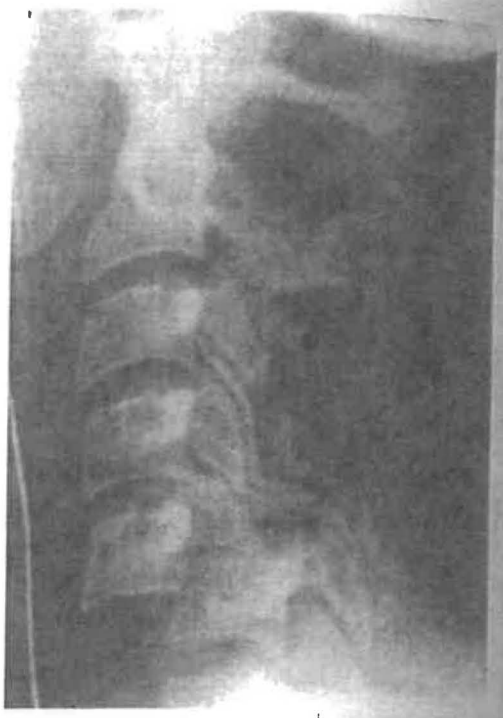

Figure 51-7. Plain lateral radiograph demonstrating C5–C6 subl[uxation] secondary to bilateral facet dislocation. (From Wilberger JE: Spinal Cord Injuries in Children. Mount Kisco, NY, Futura Publishing, 1986, p. 25.)

the spinal canal, and kyphotic angulation. The hallmark of facet dislocation is subluxation (Fig. 51-7).

The most common fracture of the thoracolumbar spine is the compression or burst fracture brought about by flexion and axial loading. The T12–L1 level is the one most frequently affected. Typical radiographical features are loss of vertebral height, retropulsion of bone fragments, and kyphotic angulation. The degree of bony disruption and spinal canal compromise is well demonstrated on CT (Fig. 51-8).

A small but definite subtype of SCI occurs without any demonstrable radiographical abnormality, including the appearance on MRI. This type is more likely to occur in children rather than adults; it is often associated with a delayed onset of symptoms after injury, and there is a very poor prognosis for neurological recovery.[72]

MRI is becoming increasingly useful in the evaluation of SCI. MRI is adequate for demonstrating most bony injury and is superior to all other modalities for exposing the soft tissue and ligaments. Additionally, MRI is the only modality that allows direct visualization of the injured cord (Fig. 51-9).

Several patterns of cord injury have been identified on MRI—transection, contusion, hemorrhage, edema—and preliminary studies suggest a correlation between initial and follow-up MRI findings and neurological prognosis.[73] When MRI is not available, not feasible, or contraindicated, intrathecally enhanced CT scan is the best alternative. Myelography can also be performed initially when movement of the patient is feasible.

MANAGEMENT. Resuscitation and stabilization of the patient's condition must be accomplished simultaneously

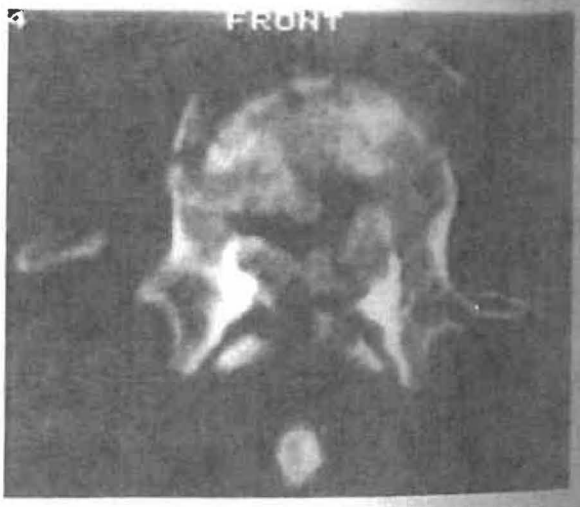

Figure 51-8. CT scan slice of an L1 compression-burst fracture with significant spinal canal compromise.

with immobilization and stabilization of the spine in the patient with SCI. Because of the frequent occurrence of multisystem injury, the principles of advanced trauma life support must be methodically applied. If significant chest or abdominal injuries coexist, immediate treatment should be directed to those injuries considered most life-threatening.

In the initial management, establishment and maintenance of the airway take precedence. With known or suspected SCI, neck manipulation should be minimized

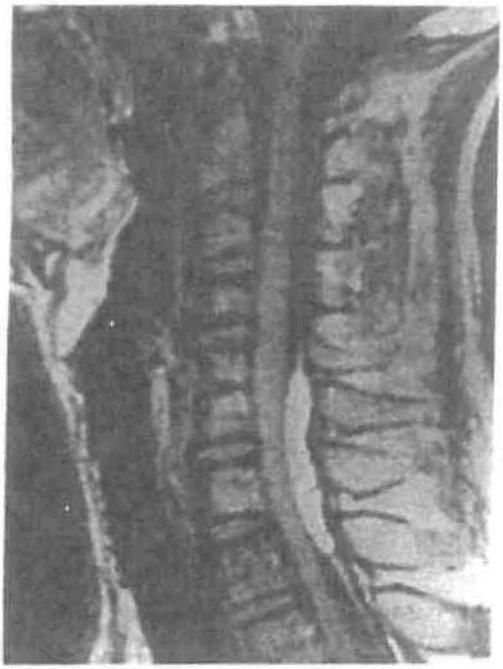

Figure 51-9. Sagittal MRI image showing a large dorsal epidural hematoma after posterior element fracture.

Appropriate large-bore intravenous lines should be placed and fluid resuscitation provided. If possible, both a Foley catheter and a nasogastric tube should be placed. When SCI is related to sports injury, the sensational nature of these cases often generates considerable interest. When these injuries do occur it is important to immobilize the athlete on the field and to leave intact any protective headgear, unless airway maintenance is threatened.

During the process of general stabilization, the clinician must remain constantly aware of the principles of spine stabilization. As noted earlier, intubation when necessary should be accomplished with the neck in as neutral position as possible. Adequate stabilization of the cervical spine requires both a hard cervical collar to prevent flexion or extension and side supports to prevent rotation. This immobilization can be accomplished by using sandbags or bolsters on the sides of the head or by taping the head to a rigid backboard. Thoracolumbar injuries are stabilized on a backboard. It is important to remember, however, that insensate patients with SCI left on hard backboards rapidly develop decubiti.

Once the patient's condition is stabilized and appropriate diagnostic studies have been accomplished, more definitive spine stabilization can be undertaken. For cervical injuries, this involves immobilization and reduction of any dislocations, usually by skeletal traction. Many devices are currently available for this purpose; the most commonly used are Gardner-Wells tongs and halo rings. Once a traction device has been placed, weights can be added to aid in spinal realignment. It has been generally recommended that no more that 5 pounds of weight be applied for each cervical level involved (i.e., for a C5 level dislocation no more than 30 pounds should be applied). In routine clinical practice, however, especially for injuries such as bilateral facet dislocations, weights in excess of 50 pounds may be necessary to achieve reduction. Some controversy exists over how rapidly reduction should be undertaken; however, most agree that with incomplete SCI, rapid intervention is most appropriate.

When traction is applied, the patient must be continually monitored both radiographically and clinically. Overtension of the spine may cause cranial nerve deficits or neurological worsening. Muscle relaxants such as diazepam are often helpful adjuncts in reducing spasm, which may inhibit efforts at reduction. Fluoroscopy may be useful for frequent radiographical monitoring during active attempts at reduction through traction. If closed reduction through traction fails, a decision must be made about open reduction and surgical stabilization. Although there are no good scientific data to indicate any definite advantages with respect to neurological improvement from rapid aggressive surgical intervention in these cases, there is a growing tendency to advocate such an approach.[74–77]

Generally, realignment of the thoracolumbar spine cannot be accomplished with external traction. Affected patients are immobilized on rotating beds until a decision is made regarding surgical intervention.

The best current initial medical and pharmacological therapy of SCI is based on the potentially reversible pathophysiological changes that occur in relation to spinal cord blood flow and the beneficial effects that have been demonstrated with high-dose methylprednisolone treatment.

Because of the known tendency for spinal cord perfusion to fall abruptly after SCI, systemic blood pressure should be vigorously supported and on occasion mild hypertension induced to ensure adequate perfusion for at least the first 24 hours after injury.[78] Fluid resuscitation should consist of an appropriate combination of crystalloid and colloid or blood replacement, if necessary. In most instances, lactated Ringer's solution or normal saline is the best initial fluid. If, despite adequate fluid replacement, the blood pressure cannot be normalized, pressors such as dopamine or phenylephrine hydrochloride (Neo-Synephrine) may be instituted. An additional temporary measure to improve peripheral vascular resistance may be a pneumatic antishock garment.

Because spinal cord blood flow is dependent not only on perfusion pressure, but also on blood rheology, it is important to consider measures to reduce blood viscosity, thereby increasing perfusion. Optimal viscosity for this purpose is achieved with a blood hematocrit in the range of 33% to 37%.

A wide variety of hemodynamic derangements is seen in association with SCI, and there is some indication that their correction improves the chances for neurological recovery. Thus, placement of a Swan-Ganz catheter is appropriate after SCI to better detect and treat these problems.

If treatment is initiated within 8 hours of injury, methylprednisolone can be given as an intravenous bolus of 30 mg/kg followed by continuous infusion of 5.4 mg/kg/hour. If treatment is initiated within 3 hours of injury, the continuous infusion should be continued for 24 hours. Patients initiating treatment 3 to 8 hours after injury should have the continuous infusion for 48 hours. The odds of a methylprednisolone-treated patient improving motor

function is greater than 2:1 when compared with other pharmacological therapies, whereas the odds of improving sensory function is 3:1.[79,80] Methlyprednisolone should not be given if more than 8 hours have elapsed since the SCI, because patients treated in this manner have a slightly worse outcome. Recently, considerable controversy has arisen over the scientific validity of the studies showing the benefits of methlyprednisolone, and the clinician should weigh the potential benefits versus the possible risks of this treatment. In patients treated for 48 hours one must be particularly vigilent for infectious complications and gastrointestinal bleeding.

The only other pharmacological treatment of SCI that has demonstrated potential benefit is GM_1 ganglioside (SYGEN). When given after methylprednisolone on a daily basis for up to 2 months following injury, this therapy increases the rate of neurological recovery. Ultimately, however, the final degree of functional improvement was no different than the level attained by placebo treatment.[81]

Once the immediate problems attendant on the SCI have been stabilized, attention must be directed to preventing the many complications—pulmonary, cardiovascular, urinary tract, gastrointestinal, skin—that typically threaten in the first week to 10 days after SCI (Fig. 51-10).

Pulmonary problems are the single most common cause of morbidity and death following SCI. The higher the anatomical level of injury, the greater the risk of neurological problems. Impaired deep breathing and coughing, on the basis of either pain or neurological compromise, increase the risks of atelectasis and pneumonia. Additionally, the need for maintaining recumbency to allow for immobilization of the spinal injury further compromises respiratory status.[82] Careful serial observation must be maintained to guard against the insidious development of respiratory insufficiency. Measurements of arterial blood gases or pulse oximetry should be supplemented by measurements of vital capacity at regular intervals. Pulmonary toilet is enhanced by regular nasotracheal suctioning, frequent chest physiotherapy, and the use of rotating beds or frames.[83]

Several cardiovascular problems may complicate acute management—vasogenic spinal shock, paroxysmal hypertension, arrhythmias, and thermoregulatory dysfunction. Vasogenic spinal shock results from peripheral pooling of blood from loss of sympathetic vasomotor control and generally requires treatment with vasopressors. The most common arrhythmia is bradycardia, which may be so pronounced as to produce hypotension. On occasion, temporary pacing may be required. Thus, all patients with SCI should be monitored hemodynamically for the first several days after injury.

The gastrointestinal (GI) tract must be monitored for the development of paralytic ileus, which almost universally occurs. Associated abdominal distention may adversely affect respiratory function. Judicious use of nasogastric suctioning should prevent these problems. Once the ileus has resolved, it is important to initiate and maintain regimens of bowel regulation and retraining to prevent constipation and impaction and their undesirable effects.

The primary source of infection after SCI is the genitourinary tract. The bladder can also account for significant long-term disabilities consequent to associated renal damage if appropriate care is not taken early on. Because reflex micturition almost always begins within several days of injury, long-term Foley catheterization is discouraged in favor of intermittent catheterization. Prophylactic antibiotics are not indicated, and bladder re-education should begin promptly.

Attention to skin care is vitally important. Immobility and lack of sensation predispose to skin breakdown. Prolonged pressure over bony prominences must be avoided. Pressure reduction can only be accomplished by frequent turning.[84]

Considerable disagreement exists regarding the place of surgery after SCI and, should surgical treatment be decided upon, what should be done and when.

General agreement exists as to the necessity for immediate immobilization and early stabilization of fractures and dislocations of the spine. Immobilization and early stabilization can be accomplished without surgical intervention (i.e., with external orthoses such as the halo) in over 60% of SCI cases. The timing and method of internal surgical stabilization are points of controversy. The single widely accepted indication for acute surgical intervention is documented neurological deterioration in association with ongoing spinal cord compression from bone and disc fragments, hematoma, or unreduced subluxation. Nevertheless, the presence of an incomplete SCI with persistent spinal cord deformity and presence of complete SCI when the perceived possibility exists for recovering some neurological function have been advocated as rationales for early aggressive surgical intervention.[12–14]

Oral and injectable diazepam, baclofen, dantrolene, and clonidine are helpful for the treatment of spasticity. When these medications are inadequate, other treatments include motor point blocks with phenol, botulinum toxin injections, and intrathecal perfusion with baclofen or morphine. Central deafferentation pain may respond to treatment with tricyclic antidepressants, valproic acid, carbamazepine,

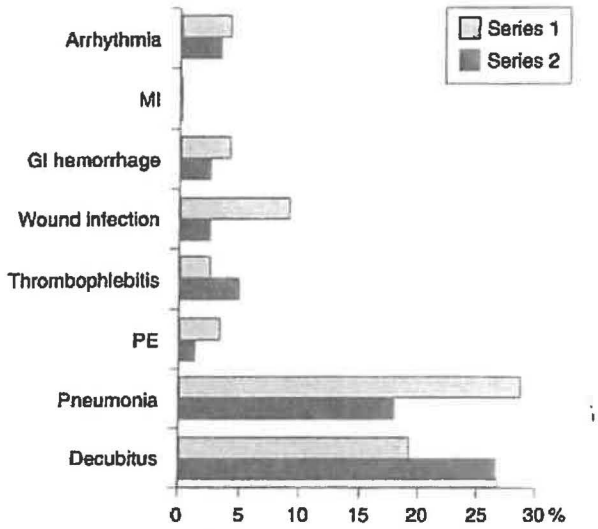

Figure 51-10. Frequency of acute complications after spinal cord injury. MI, myocardial infarction; PE, pulmonary embolus. (From American Spinal Injury Association: Standards for Neurological and Functional Classification of Spinal Cord Injury, Revised 1992, p 278.)

mexiletine, and intrathecal morphine. Occasional patients may be helped by surgical approaches such as dorsal root entry zone lesions and implantation of a dorsal column stimulator.

PROGNOSIS AND FUTURE PERSPECTIVES. It is important to attempt to quantify the natural history of the degree and extent and time course of neurological recovery after SCI to prognosticate function, determine the effectiveness of various interventions, and develop a comprehensive rehabilitation plan.

Waters and coworkers[85] reported a study of 148 patients with complete SCI followed for 5 years with annual examinations using the ASIA scoring system. Very little, if any, significant recovery was documented after 1 year of follow-up. In these patients, however, 18% had at least one level of improvement at 1 year in their neurological level of injury and 7% had two levels of improvement. Ditunno and coworkers[86] undertook a study to determine the recovery of key muscles of the upper extremities in motor-complete quadriplegics. One hundred fifty patients were followed for 24 months. Recovery persisted in muscles with no initial motor power from the ninth to twenty-fourth months, with ultimately 64% achieving some increased function. The recent National Acute Spinal Cord Injury Study found a plateauing of recovery between 6 and 12 months after injury.[83,84] If an athlete suffers an SCI, return to any level of competition is medically precluded. Risks to the individual from other less severe spinal injuries are debated, however. In general, any injury that necessitates internal surgical spinal stabilization probably obviates return to contact sports. More minor injuries that heal correctly with bracing may not limit athletic involvement.[87]

WHIPLASH INJURIES

The term *whiplash* refers to the mechanism of the neck injury, which can result from hyperextension followed by flexion that occurs when an occupant of a motor vehicle is hit from behind by another vehicle. Some clinicians use the term to also describe other types of collisions wherein the neck is subjected to different sequences and combinations of flexion, extension, and lateral motion. The term was first used in 1928. Other terms used include cervical sprain, cervical myofascial pain syndrome, acceleration-deceleration injury, and hyperextension injury.[88,89]

PATHOGENESIS AND PATHOPHYSIOLOGY. Animal and human studies have demonstrated structural damage, including muscle tears, rupture of ligaments, avulsions and herniations of intervertebral discs, retropharyngeal hematoma, nerve root damage, cervical sympathetic chain injury, and hemarthrosis of facet joints.

EPIDEMIOLOGY AND RISK FACTORS. In 2004, there were 10,900,000 motor vehicle accidents, including 3,330,000 rear-end collisions, in the United States. Although neck injuries can commonly occur after side or front impact collisions, rear-end collisions are responsible for about 85% of all whiplash injuries. More than 1 million people sustain whiplash-type injuries per year in the United States. Women have persistent neck pain more often than men, especially women in the 20- to 40-year age group, by a ratio of 7:3. The greater susceptibility of women to whiplash injuries may be due to a narrower neck with less muscle mass supporting a head of roughly the same volume compared with men.

CLINICAL FEATURES AND ASSOCIATED DISORDERS. Following a motor vehicle accident with a whiplash-type mechanism of injury, 62% of patients presenting to the emergency room complain of pain. The onset of pain is within 24 hours in 93% of the injured. The most common cause of the neck pain is myofascial injury. Cervical disc herniations, cervical spine fractures, and dislocations are uncommon. Cervical facet joint injury can also be a source of pain.[90]

Eighty percent of patients with whiplash-type injuries complain of headaches during the first 4 weeks after the accident. The headaches are usually of the muscle contraction type and are often associated with greater occipital neuralgia. Whiplash trauma can also injure the temporomandibular joint and can cause jaw pain often associated with headache. Headache may be referred from the C2–C3 facet joint that is innervated by the third occipital nerve. Occasionally, whiplash injuries can precipitate new migraine with recurring episodes including classic migraine with aura, migraine attacks without aura, and basilar migraine.

Dizziness is a common complaint and can be due to dysfunction of the vestibular apparatus, cervical proprioceptive system, brain stem, and cervical sympathetic nerves.

Paresthesias of the upper extremities can be referred from trigger points, brachial plexopathy, facet joint disease, entrapment neuropathies, cervical radiculopathy, and spinal cord compression. Thoracic outlet syndrome, which occurs four times more often in women than men, is a common cause of paresthesias, which often radiate down the ulnar arm and forearm into the fourth and fifth fingers. Eighty-five percent of these cases are of the nonneurogenic type with subjective complaints but no objective findings.

Cognitive impairment due to a whiplash injury without direct head trauma is a controversial topic with evidence for and against. [91]

Interscapular pain and low back pain are frequent complaints after whiplash injuries and are reported in 20% and 35% of patients, respectively, after the injury. Visual symptoms, especially blurred vision, are often reported by patients and are usually due to convergence insufficiency, although oculomotor palsies can occasionally occur (Video 32, Cranial Nerve III Palsy). Rare sequelae include cervical dystonia or torticollis, transient global amnesia, esophageal perforation and descending mediastinitis, hypoglossal nerve palsy, cervical epidural hematoma, superior laryngeal nerve palsy, and internal carotid and vertebral artery dissections (Video 52, Torticollis).

DIFFERENTIAL DIAGNOSIS. Whenever patients have pain complaints without objective findings, nonorganic explanations should be considered. Other possible causes of pain complaints after whiplash injuries include psychological problems, secondary gain and malingering in cases in which litigation is pending, and social and peer copying.[92,93]

EVALUATION. A cervical spine MRI study is helpful to evaluate patients with persistent complaints of neck pain and paresthesias or those with abnormal results on examination. When radiographical abnormalities are present, the possibility that they are pre-existent should be considered.

Cervical spondylosis, degenerative disc disease, and cervical disc protrusions occur with increasing frequency with older age and are often asymptomatic.

MANAGEMENT. Neck pain is often treated initially with ice, then with heat, nonsteroidal anti-inflammatory drugs, muscle relaxants, and pain medications. Use of soft cervical collars should be kept to a minimum during the first 2 to 3 weeks and then avoided. Range-of-motion exercises, physical therapy, trigger point injections, and TENS units may be helpful for patients with persistent complaints. Percutaneous radiofrequency neurotomy may be beneficial for the treatment of chronic facet joint pain documented by anesthetic blocks.[94]

PROGNOSIS AND FUTURE PERSPECTIVES. Neck pain and headaches can persist for months and sometimes years in a minority of patients. According to various studies, neck pain and headache, respectively, have been reported as persisting for the following intervals after the accident[95,96]: 1 month, 64% and 82%; 3 months, 38% and 35%; 1 year, 19% and 21%; and 2 years, 16% and 15%. Psychosocial factors and personality traits are not predictive of the duration of symptoms. Risk factors for more severe symptoms include older age, female gender, a rear-end collision, and a rotated or inclined head position at the moment of impact. Despite the common belief that pending litigation is responsible for persistent symptoms, litigants and nonlitigants have similar psychological profiles and similar recovery rates. Most patients who are still symptomatic at the time when litigation is settled are not cured by a verdict.

BRACHIAL AND LUMBOSACRAL PLEXOPATHIES

PATHOGENESIS AND PATHOPHYSIOLOGY. Trauma can result in variable injury to the myelin, axon, and connective tissue covering of plexus elements. Mild injuries may result in focal demyelination with conduction slowing. More severe injuries may cause demyelinating conduction block (neurapraxia), which can occur alone or in association with axon degeneration. Axonotmesis, which is loss of the relative continuity of the axon and myelin but preservation of the connective tissue framework, leads to wallerian degeneration. *Neurotmesis* refers to an injury in which the axons and investing connective tissues lose their continuity. A combination of different grades of injury can occur. An axonal nerve injury in continuity may heal as a neuroma that consists of axons growing in all directions surrounded by connective tissue. Root avulsion is the traumatic separation of a nerve root from the spinal cord.[97]

EPIDEMIOLOGY AND RISK FACTORS. Closed lesions usually resulting from traction are the most common causes of traumatic brachial plexopathy. Stretch injuries are responsible for about 70% of serious brachial plexus injuries. High-velocity closed injuries, which usually involve the supraclavicular plexus, are primarily due to motor vehicle accidents, occupational injuries, and falls. Falls, which result in scapular and proximal humeral fractures and humeral head dislocations, are the most common causes of infraclavicular injuries. Brachial plexus injury, usually involving the lower trunk or medial cord, occurs in 5% of patients undergoing coronary artery bypass surgery. Brachial plexus injury is probably due to traction associated with sternal retraction.[98] Most brachial plexus avulsion injuries are due to motor vehicle accidents, especially motorcycle accidents; industrial accidents; obstetrical injury; falls; objects falling on the shoulder; and sports injuries (most commonly football, bicycling, skiing, and equestrian activities). Root avulsions more commonly involve the C7, C8, and T1 roots, whereas extraforaminal ruptures more commonly affect the C5 and C6 roots.

The less common open lesions, which most often affect the infraclavicular plexus, are often associated with injuries to major limb vessels and the lung. Gunshot wounds, which account for 25% of the cases of surgical repair of the brachial plexus in the United States, usually involve the infraclavicular or retroclavicular plexus and less often the supraclavicular plexus. Laceration injuries of the brachial plexus are usually the result of broken glass and knives.

Injury to the lumbosacral plexus is uncommon because it is protected by the pelvic bony ring. Pelvic fractures, which are usually due to motor vehicle accidents, crush injuries, and falls from a height, result in lumbosacral plexus injury in about 7% of cases.

CLINICAL FEATURES AND ASSOCIATED DISORDERS. Plexus injuries can result in sensory loss, paresthesias, motor loss, and pain, which can be particularly severe after root avulsions. A detailed knowledge of the anatomy of the brachial and lumbosacral plexus is essential to localizing lesions (see Chapters 15, 19, and 20).

Burners and *stingers* are synonymous terms that refer to the radicular and plexus symptoms following sudden depression of the shoulder in contact sports, usually football. The player experiences burning dysesthesias going down the ipsilateral upper extremity, often into the thumb, and sometimes weakness of the biceps and shoulder girdle muscles. The symptoms usually resolve within a few minutes, although occasional cases last for weeks.

Other causes of acute compression of the brachial plexus include the following: prolonged compression in someone inebriated or comatose; compression of the plexus against the first rib due to heavy backpacks carried by students or soldiers; prolonged firing of shotguns or rifles; and shoulder restraints in motor vehicles. In addition to coronary artery bypass surgery, other intraoperative causes of plexus injury include malpositioning of the arm, often affecting the upper plexus; damage during other procedures such as radical mastectomies, transaxillary arterial by passes, and biopsies; orthopedic procedures on the humerus or shoulder; and ironically, first rib resections and scalenotomies performed to treat thoracic outlet syndrome.[98] False aneurysms of the axillary artery due to trauma and hematomas from transaxillary percutaneous angiograms can also injure elements of the plexus. Stretch injuries of the plexus in neonates, often resulting from shoulder dystocia, most commonly involve the upper trunk (Erb's palsy) and only occasionally the lower trunk (Klumpke's palsy).

Repetitive activity such as working at a keyboard or playing a musical instrument and single injuries such as a fall or whiplash-type injury commonly result in the nonneurogenic type of thoracic outlet syndrome. Patients may complain of aching of the shoulder and arm with paresthesias going down the arm into the fourth and fifth fingers. The symptoms may be worse at night, with

repetitive activity, or with use of the arm overhead. Although they are nonspecific causes, medial supraclavicular palpation and the exaggerated military posture may reproduce the symptoms. Most of the patients are women; long necks, droopy shoulders, and pendulous breasts may be contributing factors. No objective findings appear on examination or electrodiagnostic studies.[97]

In addition to pelvic fractures and gunshot wounds, there are other important causes of lumbosacral plexus injury. During labor and delivery, pressure on the lumbosacral trunk by the fetal head or forceps may result in a postpartum footdrop. Ischemic injury of the plexus can result from inadvertent injection of drugs into the gluteal arteries, leading to a propagating thrombus to the iliac arteries. Blunt trauma to the abdomen can rarely result in a retroperitoneal hemorrhage into the psoas muscle, which compresses the lumbar plexus, causing pain and extremity weakness. Neuromas may form after partial or complete injuries to peripheral nerves. The only neuromas that are symptomatic are those that contain sensory nerve fibers. Sensory nerves of the hands and feet are especially likely to form painful neuromas. Symptomatic neuromas produce a burning or aching pain that may radiate proximally or distally. Tinel's sign may be present. The diagnosis can be confirmed by temporary relief of symptoms after injection of local anesthetic. Injection of a corticosteroid may significantly improve or cure symptoms in some cases. Various surgical approaches such as nerve grafting, decompression and translocation, excisional neurectomy, and transposition may be appropriate, depending on the case.[99]

DIFFERENTIAL DIAGNOSIS AND EVALUATION. Cervical and lumbosacral disc herniations and peripheral nerve injuries should be considered. The clinical examination can often localize the lesion as pre- or postganglionic or to various elements of the plexus. For example, the presence of Horner's syndrome indicates lesions of the roots of C8 and T1 proximal to the site where the white rami communicans exits to enter the sympathetic chain. Depending on the specific injury, various imaging techniques including plain radiographs, CT and MRI scans, arteriography, venography, and myelography may be indicated.[100] Pseudomeningoceles, which are formed when a nerve root is avulsed and the meninges are pulled through the neural foramina, may be detected on myelography, followed by CT scan as well as MRI. Electromyography (EMG) and nerve conduction studies can be helpful in localizing and determining the extent of the lesion.

MANAGEMENT. Management depends on the location of the lesions, the grade of nerve injury, and the presence of root avulsions. Patients with acute transections of the brachial plexus due to lacerations with a knife or glass should undergo relatively rapid primary repair. For closed stretch injuries with severe axonal degeneration present on electrophysiological studies 3 to 5 months after the injury, surgical exploration and repair are indicated.[101] Because missile wounds usually leave the nerve in continuity, initial management is often conservative. Although avulsion of nerve roots has been regarded as an untreatable injury, animal and experimental human studies suggest that implantation of ventral roots into the spinal cord may lead to recovery of motor function especially with use of neurotrophic factors.[102–104]

Up to 80% of patients with obstetrical palsy have no paresis or mild paresis by the age of 1 year. Surgery for infants with obstetrical palsy not improving is controversial, with recommendations for the time of surgery ranging from 3 to 9 months of age. Non-neurogenic thoracic outlet syndrome may respond to Peet's exercises, which strengthen the muscles of the pectoral girdle and improve posture. In very occasional cases, scalenotomy or first rib resection may be indicated. Physical and surgical rehabilitation may help to improve function after brachial plexus injuries.

Pain following brachial plexus injuries may be difficult to manage with various treatments depending upon the type and severity of the pain. Treatments range from pain pills, anticonvulsants, and TENS units to stellate ganglion blocks and sympathectomy, dorsal column stimulators, and dorsal root entry zone lesions. Similar considerations apply to lumbosacral plexus injuries. When indicated, surgery may be helpful in some cases of pelvic fractures, gunshot wounds, stab wounds or lacerations, and iatrogenic injuries.

PROGNOSIS AND FUTURE PERSPECTIVES. In cases of neurapraxia, spontaneous recovery may occur within 3 to 4 months of the injury. Because of the long regeneration distances and lack of collateral sprouting, the intrinsic hand muscles with lower trunk lesions and the muscles below the knee with sacral plexopathies reinnervate poorly after severe axon loss lesions. For severe brachial plexus lesions, satisfactory results may occur postoperatively in up to 70% of patients after primary repair and up to 48% after a nerve graft. Fifty pecent to 85% of patients with non-neurogenic thoracic outlet syndrome may improve with exercises.[103]

COMPLEX REGIONAL PAIN SYNDROME (REFLEX SYMPATHETIC DYSTROPHY AND CAUSALGIA)

In 1864, the Philadelphia neurologist S. Wier Mitchell and colleagues reported the cases of Union soldiers who sustained gunshot wounds at the battle of Gettysburg resulting in partial nerve lesions of the brachial plexus and peripheral nerves followed by a severe burning pain of the hand or foot associated with trophic changes.[105] Mitchell coined the term *causalgia*, which is from the Greek for "burning pain," to describe this syndrome. In 1995, a consensus development conference grouped these disorders under the term complex regional pain syndrome (CRPS) type I (corresponding to reflex sympathetic dystrophy) and type II (formerly termed causalgia).[106]

PATHOGENESIS AND PATHOPHYSIOLOGY. The pathophysiology of CRPS is poorly understood. Various mechanisms proposed include the following: sympathetic hyperactivity, ephaptic transmission between sympathetic and primary afferents, a reverberating circuit in the spinal internuncial pool, sensitization of wide dynamic range neurons in the dorsal horn, and neurogenic inflammation. Despite common perception and former nomenclature, some researchers strongly argue that the sympathetic nervous system is not involved.[107]

EPIDEMIOLOGY AND RISK FACTORS. CRPS II may occur in up to 5% of patients after partial peripheral nerve

injury, but it does not occur when a nerve is completely severed. The incidence of CRPS I is 1% to 2% after various fractures but is particularly common following Colles' fracture, occurring in about 30%. CRPS I may occur after various other injuries, including minor soft tissue trauma, operations such as carpal tunnel release and arthroscopic knee surgery, myocardial and cerebral infarction, and frostbite and burns. In up to 25% of CRPS I cases, a precipitant cause is not identified. CRPS I occurs in all ages with a median of 40 years. Females account for about 70% of cases.

CLINICAL FEATURES AND ASSOCIATED DISORDERS. CRPS I is a syndrome with variable major components of burning pain, autonomic dysfunction, edema, dystrophy and atrophy, and sometimes a movement disorder.[108] When this pain syndrome follows peripheral nerve injury, the term *CRPS II* is used. Various other terms for CRPS I are used in addition to reflex sympathetic dystrophy including Sudeck's atrophy, algodystrophy, post-traumatic dystrophy, reflex neurovascular dystrophy, shoulder-hand syndrome, and sympathetic maintained pain syndrome.

The symptoms of CRPS may develop within days to months after injury. The pain, which begins in the area of the injury or event, may then spread throughout the extremity and, in perhaps 5% of cases, involves the contralateral extremity (rarely, three and four extremities may become involved). The burning, aching, or constant pain is associated with hyperalgesia (lower pain threshold with enhanced pain perception), allodynia (pain induced by innocuous mechanical and thermal stimuli, especially cold), and hyperpathia (an exaggerated response to a stimulus, which may be delayed and may persist after cessation of the stimulus). Spontaneous paroxysmal shooting, sharp, or electrical sensations may be described. Decreased sensation may be present in affected areas. Proprioception may be limited. As in other pain disorders, CRPS I patients are often depressed and anxious.

Edema over the site of injury and distally may be present. At first, the involved extremity is usually warm, red, and dry but occasionally may be cold. Over time, the extremity usually becomes cool, pale, cyanotic, and hyperhydrotic. Early on, the nails may become thickened and the hair darker. Later, the hair may be lost in the affected areas, the skin may become shiny, and the nails may break. Dystrophy and atrophy of subcutaneous tissue, muscles, and bone may be present. Nodular fasciitis of the palmar or plantar skin may be present. CRPS I affecting the hand may be associated with a frozen shoulder.[109] Motor manifestations variably present include weakness, an enhanced physiological tremor, spasm and increased reflexes, focal dystonia, and an inability to initiate movement. Exercise or use may aggravate CRPS I complaints.

DIFFERENTIAL DIAGNOSIS. Other disorders that may have some features of CRPS I include chronic arterial insufficiency, thrombophlebitis, infection, collagen vascular disorders, postherpetic neuralgia, entrapment neuropathies (which can coexist with CRPS I), painful neuromas, plexopathies and radiculopathies, phantom limb pain, and central pain syndromes. When pain complaints are the major problem, psychological causes such as depression, factitious disorders, conversion, and malingering should be considered.

After amputation of an extremity, up to 85% of patients describe phantom limb pain, which may still be present in about 30% 1 year later. The patients describe pain, which may be a continuous numbing or an intermittent sharp, stabbing, shooting, cramping, or burning sensation, as being present either in the stump or in the absent limb. Both peripheral and central mechanisms for the pain have been suggested, with one study showing evidence of plastic changes in the primary somatosensory cortex.[110] Despite various medical and surgical treatments, the pain is often intractable.

EVALUATION AND MANAGEMENT. The diagnosis of CRPS is based on the symptoms and signs. Laboratory testing sometimes used includes thermography and a triple-phase bone scan (which may reveal abnormal uptake and increased periarticular uptake in 30% of patients); abnormal findings are not specific. Pain relief following sympathetic blockade or an intravenous phentolamine test is supportive of the diagnosis. The possibility of a placebo response should be considered.

The inciting disorder should be appropriately diagnosed and treated. Although many treatments have been proposed, there is a lack of prospective and controlled studies with controversy over inclusion criteria. Medications reported as useful include the following: narcotic and nonsteroidal anti-inflammatory drugs; adrenergic blockers such as clonidine and prazosin; tricyclic antidepressants; anticonvulsants such as carbamazepine and gabapentin; corticosteroids; topical DMSO cream; and intranasal calcitonin. Physical therapy modalities, rehabilitation, TENS units, and psychological treatment are often worth while. A series of five sympathetic anesthetic blocks of the stellate ganglion for upper extremity or lumbar chain for lower extremity cases can be beneficial. If the blocks improve the pain but only temporarily, many clinicians recommend sympathectomy. Dorsal column stimulators and morphine intrathecal pumps are sometimes recommended for refractory cases. Some patients with dystonia associated with reflex sympathetic dystrophy may respond to treatment with intrathecal baclofen. Recently spinal cord stimulation has been successfully used in the management of CRPS. These electrodes implanted over the posterior columns relieve pain, thereby allowing physical therapy, and activity to break the cycle of disuse.[111] Spinal cord stimulators were initially inspired by the gate control theory but their action clearly involves more than just modulation in the dorsal horns. While the exact mechanisms of pain relief by spinal cord stimulation are not known, the following have been proposed:

1. Orthodromic stimulation causing supraspinal pain modulation
2. Modulation of activity in the sympathetic system
3. Release of neuromodulators within the brain or spinal cord
4. Inhibition of transmission in the spinothalamic tract
5. Inhibition of C fibers in the substantia gelatinosa by antidromic conduction through A-β fibers

PROGNOSIS AND FUTURE PERSPECTIVES. Prognostic information is quite limited. Many clinicians believe that recovery occurs more frequently when treatment is begun early in the course. Pain may still be present in 25% of cases with CRPS I after 1 year and in 60% of patients with CRPS II after 3 years. Reported response rates for surgical

sympathectomy range from 12% to 97%. RSD may resolve early in the clinical course only to return weeks or months later.

MOUNTAIN SICKNESS

PATHOGENESIS AND PATHOPHYSIOLOGY. As the partial pressure of oxygen decreases with increasing altitude, ventilation rises, leading to respiratory alkalosis. Although hypocapnia alone results in cerebral vasoconstriction, hypoxia produces a net decline of cerebral vascular resistance and increased cerebral blood flow. Hypoxia can result in cerebral edema, which may be due to cerebral vasodilatation and elevated cerebral capillary hydrostatic pressure. An increase in sympathetic activity follows, causing an elevated heart rate, pulmonary vasoconstriction, and an initial increase and later decrease of cerebral blood flow.[112]

EPIDEMIOLOGY AND RISK FACTORS. Acute mountain sickness (AMS) develops in about 25% of visitors to moderate altitudes (6300 to 9700 feet). About 100 million tourists visit altitudes above 2000 meters worldwide per year. Symptoms usually occur within the first 12 hours of arrival but may be delayed 24 hours or more. Risk factors for AMS include those who are younger than 60 years, are less physically fit, live at sea level, have a history of AMS, are obese or female, or have underlying lung problems.[113] AMS develops into high-altitude cerebral edema (HACE) in perhaps 1.5% of cases. Alcohol use inhibits acute ventilatory adaptation to mild hypoxia at moderate altitude and may be a risk factor.

CLINICAL FEATURES AND ASSOCIATED DISORDERS. AMS, which usually occurs above 8200 feet, is defined by the presence of a headache and at least one of the following symptoms: gastrointestinal upset (anorexia, nausea, or vomiting), fatigue or weakness, dizziness or lightheadedness, and difficulty sleeping.[114] The headache is usually bilateral but may be unilateral. HACE, which is uncommon below 12,000 feet, is defined by the presence of a change in mental status or ataxia in a person with AMS or the presence of both mental status change and ataxia in a person without AMS. HACE, which can lead to coma and death, may be associated with urinary incontinence, papilledema, cranial nerve palsies, tremor, and abnormalities in limb tone (Video 1, Cranial Nerve VI Palsy; Video 214, Stance Ataxia).

High-altitude pulmonary edema (HAPE), which can occur along with HACE, usually occurs above 9840 feet. High-altitude retinal hemorrhage is common in people who go above 15,000 feet.

EVALUATION AND MANAGEMENT. The diagnosis is usually a clinical one that is made in the setting of a recent altitude ascent. Altitude sickness may be prevented in those planning higher ascents by starting below 8000 feet, resting the first day, and then ascending about 1000 feet per day. Sleeping at a lower altitude at night may be helpful because hypoxia is worse with sleep. It is also important for climbers to keep well hydrated and avoid alcohol. Acetazolamide, at a dose of 250 to 500 mg twice daily starting 24 hours before ascent and continuing for 2 days, may prevent AMS and improve sleep. Other medications which may reduce the risk and symptoms of AMS include the following: aspirin 320 mg 1 hour before arrival at high altitude and then one dose every 4 hours for two doses (for a total of three doses)[115]; and dexamethasone 2 mg every 6 hours or 4 mg every 12 hours orally starting 24 to 48 hours before arrival at high altitude and continuing for several days.

AMS may be treated symptomatically with rest, mild analgesics, alcohol avoidance, and adequate hydration. Depending upon the severity of symptoms, avoiding going any higher or slowing the rate of ascent or descent may be necessary. Acetazolamide 250 mg orally twice a day may also help acute symptoms. For HACE, immediate descent should be initiated if possible, and, if not, HACE may be treated with the use of a portable hyperbaric bag (2 to 4 psi for a minimum of 2 hours), administration of oxygen (2 to 4 L/minute), and dexamethasone (8 mg orally, IM, or IV initially and then 4 mg every 6 hours). Acetazolamide should be given in instances when descent or evacuation is delayed.

PROGNOSIS AND FUTURE PERSPECTIVES. AMS usually resolves after 16 to 72 hours at altitude. When descent is impossible and treatment is unavailable, HACE and HAPE have a mortality rate up to 50%.

DECOMPRESSION SICKNESS

PATHOGENESIS AND PATHOPHYSIOLOGY. With descent, the partial pressure of the gases breathed by the diver increases proportionately, according to Dalton's law. Although oxygen is metabolized, tissues soak up inert nitrogen and become saturated. With ascent, the nitrogen moves from the tissues to the blood and is exhaled by the lungs, which is a process termed *decompression*. When ascent is too rapid and the tissues are supersaturated, dissolved gas changes to free gas, which creates bubbles. When the filtering capacity of the pulmonary capillaries is exceeded, the bubbles enter the arterial circulation. Symptoms depend on those tissues in which the bubbles accumulate. Bubbles in the paravertebral veins, Batson's plexus, can result in stasis and venous infarction in the spinal cord. Cerebral injury can develop from bubbles occluding vessels or directly disrupting tissue. Symptoms may also result from secondary effects such as activation of complement, platelet aggregation, and release of vasoactive mediators.

EPIDEMIOLOGY AND RISK FACTORS. Decompression sickness (DCS), also referred to as *the bends* or *caisson disease*, usually affects divers and caisson workers but can also occur in pilots during rapid ascent in a nonpressurized cabin. About 900 cases of DCS are reported yearly in the United States among recreational scuba divers. Most accidents occur in inexperienced divers. The incidence of DCS for recreational divers is 1 per 5000 to 10,000 dives, and for commercial divers, one incident per 500 to 1000 dives. Up to 50% of cases of DCS occur in divers who claimed to have been diving within the limits set by a standard table or decompression computer. Divers with a patent foramen ovale are five times more likely to sustain serious decompression problems because bubbles can go directly from the right to left atrium without being filtered

by the lungs.[116] Right-to-left shunts are more common in those with migraine with aura who are more likely to suffer DCS.[117]

CLINICAL FEATURES AND ASSOCIATED DISORDERS. DCS, which can occur after diving to a depth of more than 25 feet, usually appears within a few minutes to a few hours after the end of a dive. Mild DCS (type I) is defined by pain usually in the joints (bends) and itching of the skin. Serious DCS (type II) is characterized by neurological problems. Involvement of the thoracic spinal cord, the most commonly affected area, leads to low back or pelvic pain and dysesthesias, which may be accompanied by sensory loss, weakness, and incontinence. Less often, the brain may be involved, resulting in various symptoms and signs such as headache, confusion, lethargy, vertigo, speech disturbance, hemiparesis, visual impairment, and seizures, depending upon the site of the insult (Video 4, Pronator Drift).[118]

Rupture of alveoli, which may occur when a diver ascends without venting air from the lungs or from blockage of part of the bronchial tree, can result in additional arterial gas embolism. During ascent or shortly after surfacing, divers may develop various problems ranging from acute respiratory distress and headache to cardiorespiratory arrest and seizures. Depending on the site of embolism to the brain, cortical blindness, dysphasia, and hemiplegia may occur (Video 3, Spastic Gait). Arterial gas embolism and DCS may coexist.

EVALUATION AND MANAGEMENT. DCS is typically diagnosed based on the findings on history and physical examination. The results of MRI studies of the brain and spinal cord may often be normal or may show only nonspecific abnormalities in cases of DCS.[118] Initially, high concentrations of oxygen should be administered by face mask to increase the resorption of gas bubbles. Fluid administration, ideally a hyperosmolar drink, may result in more rapid elimination of gases and treat dehydration. As soon as possible, the patient should undergo recompression in a hyperbaric chamber while breathing oxygen.[119]

PROGNOSIS AND FUTURE PERSPECTIVES. Prompt treatment usually results in a complete recovery. Neurological deficits may persist in patients in whom recompression is delayed or not done.[120]

LIGHTNING AND ELECTRICAL INJURIES

PATHOGENESIS AND PATHOPHYSIOLOGY. Although incompletely understood, the two major causes of tissue damage are thermal injury and electroporation, which is the production and expansion of transient aqueous pores in the lipid bilayer component of the cell membrane.[121]

EPIDEMIOLOGY AND RISK FACTORS. About 100 lightning- and 1500 technical electricity-related deaths per year occur in the United States. The following routes can produce lightning injury. A direct strike is the most damaging, which is more common if the person is carrying a metal conductor such as an umbrella or golf club above shoulder level. A side flash or splash injury occurs when lightning first strikes a tall object such as a tree and then arcs to the person standing next to the object or when lightning first strikes a person or animal and then the second victim. A ground or stride current occurs when lightning strikes the ground first and then travels along the surface before reaching the person. Occasionally, people can sustain injury indoors while on the telephone from current conducted through the lines or in the bath or shower from ground current traveling along the water pipes.

Technical electrical injuries can occur from exposure to high-voltage electricity (1000 volts or more) and low-voltage electricity. High-voltage electricity injuries, which are almost always work related, account for about 70% of electrical injuries and deaths. Low-voltage injuries typically happen in the home with the following common circumstances: the use of electrical appliances while standing on a wet floor or while in the bathtub, children playing with outlets or wires, or use of faulty electrical equipment. When the energized conductor is held in the hand, alternating current exposure in the range of 8 to 22 mA at 60 Hz may result in long exposure due to a state of tetanic contraction of flexor muscles of the forearm and hand, which is paradoxically termed *let go current*.[122]

CLINICAL FEATURES, AND ASSOCIATED DISORDERS. Lightning and electrical injuries may result in sudden death or cerebral hypoxia from inducing ventricular fibrillation. Transient loss of consciousness, confusion, and amnesia may also occur.[123] Lightning injury may result in keraunoparalysis, which is a transient paralysis usually of the lower extremities associated with sensory loss and pale skin. Lightning and electrical injuries may also cause acute spinal cord, focal brain, and peripheral nerve damage and rhabdomyolysis. Deep burns are more common with electrical than with lightning injuries. Delayed neurological disorders associated with lightning and electrical injuries include cognitive deficits, motor neuron disease, parkinsonism, choreoathetosis, dystonia, myoclonus, basilar artery thrombosis, seizures, myelopathy, generalized polyneuropathy, and complex regional pain syndrome.[124–126] An additional type of injury is secondary trauma, such as from falls.

MANAGEMENT, PROGNOSIS, AND FUTURE PERSPECTIVES. Evaluation and management depend on the sites, types, and extent of injury.[127] About one third of lightning strikes are fatal. The symptoms and signs of keraunoparalysis typically resolve within a few hours. Victims of lightning and electrical injuries who are comatose due to a hypoxic encephalopathy as a result of cardiac arrest usually have a poor prognosis. Most patients with spinal cord injuries due to lightning and electrical injuries have permanent disability.

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